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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Next Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

September 27, 28, 29, 1956

Atlantic City, N. J.

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Chairman, Scientific Sessions Committee for 1956

American Academy of Periodontology

Dr. Archer, long active member of the American Academy of Periodontology, is Chairman of the Scientific Sessions Committee of the American Academy of Periodontology this year. His duties include supervising the program of the meeting of the American Academy of Periodontology in Atlantic City on September 27-29. Other members of the committee include: Dr. Donald Kerr and Dr. B. O. A. Thomas.

Dr. Archer is a graduate of Chicago College of Dental Surgery, a Diplomate of the American Board of Periodontology, Vice-chairman this year of the Periodontia Section of the American Dental Association, Chairman of the Scientific Program for the Odontographic Society of Chicago, Fellow of the American College of Dentists and past President of the Alumni Association of C.C.D.S.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-SIX

The Epithelial Attachment (The Attached Epithelial Cuff)*

by BALINT J. ORBAN, M.D., D.D.S., *Colorado Springs, Colorado;* AND HARBANS BHATIA, D.D.S., M.S.; JOHN ANTON KOLLAR, JR., D.D.S.; FRANK M. WENTZ, D.D.S., M.S., PH.D.,** *Chicago, Illinois*

FOR a long time, the relation of gingival epithelium to the enamel of the tooth was considered as only one of close approximation. In this concept, it was believed that a capillary space between the gingival epithelium and enamel extended to the cemento-enamel junction from the time the tooth had erupted into the oral cavity. In 1921, Gottlieb[1] discovered that the epithelium of the gingiva is in organic connection with the enamel surface and therefore, only a shallow crevice exists at the free border of the gingiva (Fig. 1). Gottlieb called the relation between epithelium and enamel, "Epithel Ansatz"—epithelial attachment. The word "Ansatz"—attachment was used to connote a relation similar to that existing between a muscle or tendon to a bone.

While there are many problems related to our question, this paper will deal only with the central problem—namely, the existence or non-existence of an organic attachment of the epithelium to the enamel surface. The attached bibliography [2-22] covers most of these problems.

In 1952, Jens Waerhaug published a monograph, "The Gingival Pocket,"[23] in which he asserts that "The bottom of the clinical pocket was found to be situated consistently at the cemento-enamel junction." "Thus it was evident that no epithelial attachment existed." Waerhaug amplified these statements by saying that "the bottom of the clinical pocket is situated at the deepest line of epithelial attachment, thus the 'epithelial attachment' belongs to the lining of the pocket." Almost identical statements have been made by Waerhaug repeatedly in recent papers.[24, 26, 27]

An illustration from one of these papers[24] is reproduced here to illustrate Waerhaug's concept (Fig. 2). Upon a photomicrogram of a tooth and the gingiva, the outline of an instrument is superimposed†. The cutting blade of the instrument is below the apical end of calculus in the gingival pocket. However

*Paper read at the annual meeting of the American Academy of Periodontology—San Francisco—October 14, 1955.

**Loyola University School of Dentistry—Chicago, Illinois. The Colorado Dental Foundation—Colorado Springs, Colorado.

†This method of studying the relation of instruments to the surrounding tissues is excellent and was probably first used by Bodecker[25].

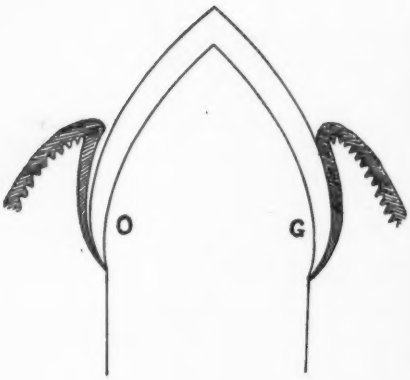


Fig. 1. Diagrammatic illustration of old concept of gingival attachment to the tooth (O), and Gottlieb's concept (G).

Fig. 2. Reproduction of Figure 1 in Waerhaug's article (ref. 24). Note that the instrument is at the point which we would consider as the bottom of the pocket. However, Waerhaug labeled the apical end of the epithelial attachment as BP, bottom of the sulcus.

Waerhaug designates the apical end of the epithelial attachment situated upon the *cementum* as the bottom of the pocket (BP).

One of Waerhaug's most important experiments was to insert into the pocket a fine steel blade, .05 mm. thick and 1 mm. wide. These blades were rounded at the angles and the edges were made smooth. They were then bent into some conformity with the curvature of the tooth surface. "This steel blade was inserted into the pocket with the greatest caution. The trifling thickness of the blade (.05 mm.) is in itself a guaranty that it cannot be pressed very hard, as it would then bend. With this technique, there was never any doubt as to where the bottom of the pocket was situated. The steel blade could always meet resistance at a certain level." "The steel blade could always very easily be inserted into the pocket to a certain level, where it met with such definite resistance that it could not be pressed any further without bending." "In all cases, it was shown to have reached the cemento-enamel junction."

In a second experiment by Waerhaug, ". . . . a lobe of gingiva was detached from the rest by means of a very sharp lancet cutting two incisions from the gingival margin a bit down below the cemento-enamel junction." Waerhaug states that by this technique, "without using any special force, the epithelium could be detached from the tooth surface all the way down to the cemento-enamel junction, where it was so firmly attached that it had to be torn loose with surgical tweezers." " . . . . the consistent result was that the clinical pocket was found to be situated at the cemento-enamel junction." Waerhaug in the legend to one of his photomicrograms, here reproduced as Figure 3, states: "Dog 6 + 6. One hour after death, steel blade inserted to bottom of clinical pocket 1.5 - 2.5 mm. below gingival margin. Distance from gingival margin to cemento-enamel junction in slide, 1.3 mm. Normal relation between 'epithelial attachment' and

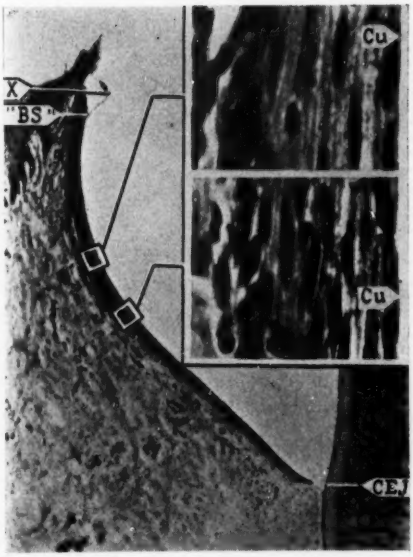


Fig. 3. Reproduction of Figure 4 of Waerhaug's article, "The Gingival Pocket" (ref. 23). Detail in text. According to Waerhaug a steel blade was inserted into the "pocket" to the cemento-enamel junction. However, there is no sign of this in this picture.

enamel to 'BS.' Higher magnification at top to the right shows somewhat irregular disposition of cells. Higher magnification below shows normal conditions."

Waerhaug remarks in the text: "in the higher magnification at the top to the right, it will be found that the epithelium is bounded outwardly by a thin membrane, Cu. The cell nuclei are narrow and elongated, and, in some places, totally missing. The intercellular spaces have a normal appearance. It cannot be decided from the section where the instrument has been inserted. It seems, however, as though the operation has brought about a certain change throughout the epithelium in this part. The higher magnification at the bottom to the right shows the state in the middle between the gingival margin and the cemento-enamel junction. Here the epithelium has the typical appearance of an 'epithelial attachment.' The cells have normal form and size with clearly visible intercellular spaces. Outwardly, it is bounded by a thin membrane, Cu, and no traces from the insertion of the steel blade can be seen."

"Since it has been shown in Figures 2, 3, and 4, that apparently normal 'epithelial attachments' may be found in spite of the gingiva having been loosened after death from the tooth surface, it seemed to be possible that the fixation and the preparation might have some significance for the final histological picture." "It was therefore evident that the fixation brought about a certain clinging between the tissue and the tooth surface." "As has been mentioned above, the soft tissue is to some extent made adherent to the primary cuticles during the fixation."

"Depths of the pocket of up to 12 mm. were often observed around erupting teeth."

Since Gottlieb's discovery meant a radical change in the concept of periodontal biology, pathology and practice, the denial of such an attachment and the return to the previous concept by Waerhaug's monograph[23] and papers[24,26,27] is of great interest. It is imperative that Waerhaug's arguments and experiments be examined most critically to avoid confusion.

MATERIALS AND METHODS

Six dogs (some young—some adult) and four adult monkeys were employed in the experimental procedures.

Three types of experiments, which will be described separately in the section on findings were conducted. They are:

I. Insertion of a steel blade into the gingival sulcus.

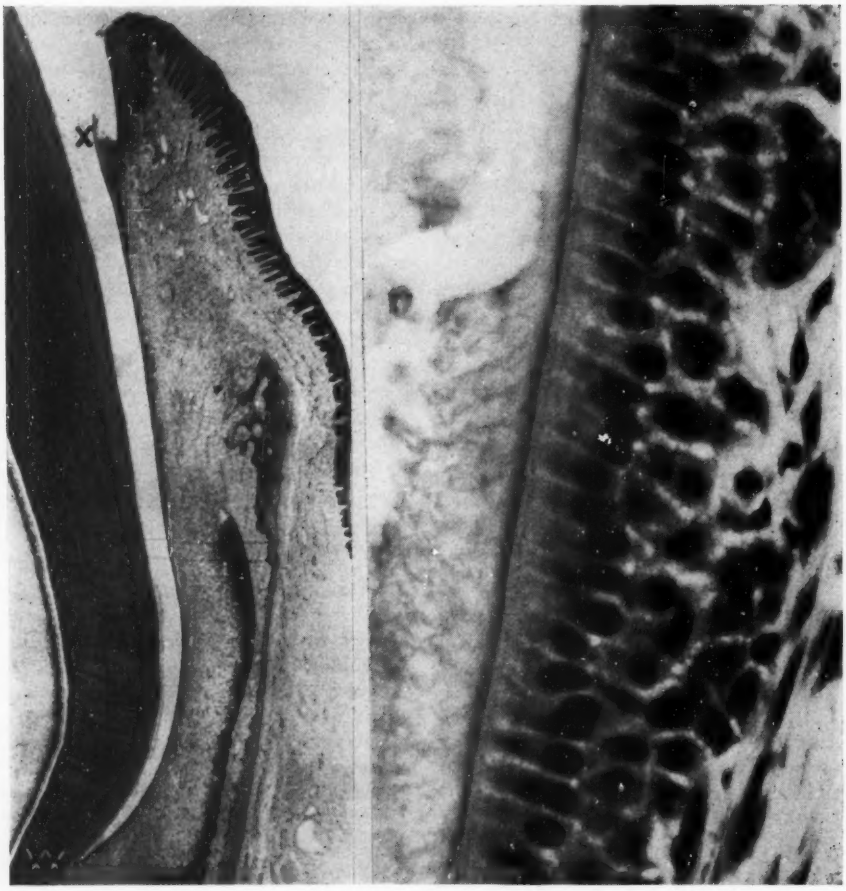
II. Gingival flap and reposition.

III. Insertion of "mylar" strip into gingival sulcus.

The operative technique and teeth used in each experiment are listed in the table.

FINDINGS

It is a common finding to see ameloblasts



A. B.

Fig. 4. Dog 1-7. Left upper fourth premolar of a young dog. Depth of sulcus, 1.5 mm. A. General View. Bottom of sulcus, X. Cemento-enamel junction, XX. The distance between the gingival margin and the cemento-enamel junction, 10 mm. The mylar film stopped at the bottom of the sulcus, X. B. Higher power view of (A) near cemento-enamel junction. Ameloblasts are observed to be attached to the organic matrix of enamel.

in union with the forming enamel matrix in the cervical areas of the crown, especially in dogs, even after eruption of the tooth (Fig. 4). This finding refutes the generalized statement made by Waerhaug that the gingival pocket reaches to the cemento-enamel junction, once the tooth has been exposed to the oral cavity, and that depths of the pocket up to 12 mm. were often observed around erupting teeth.

The findings will be listed according to the type of experiment.

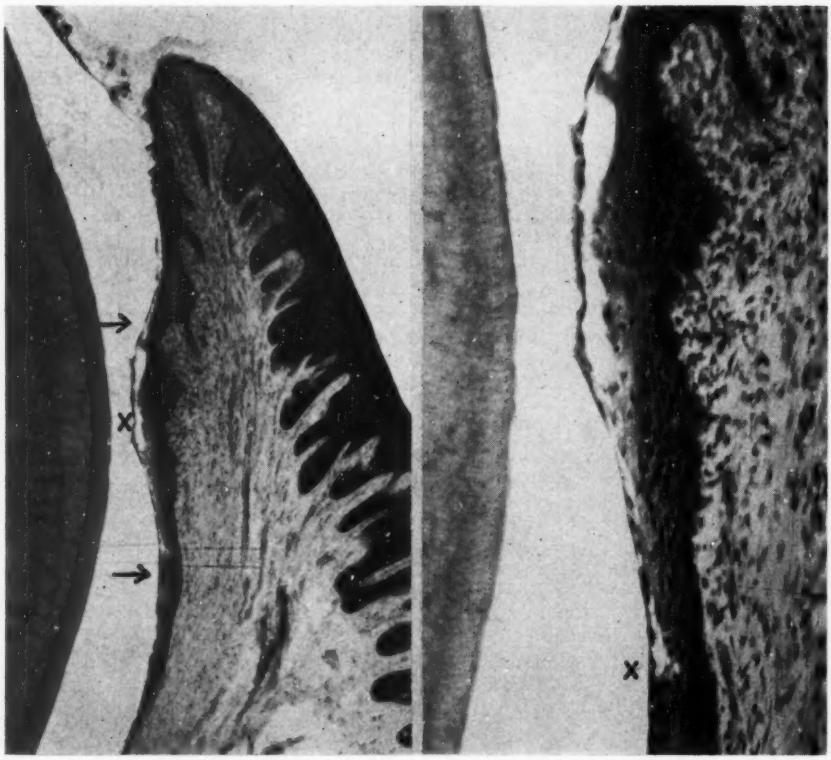
I. *Insertion of Steel Blade*

A steel blade, fashioned according to Waerhaug's specifications, was inserted into the gingival sulcus on the buccal side of a right second lower premolar of a dog, one hour after death. The blade was inserted to a depth of one millimeter, when a resistance

TABLE OF EXPERIMENTAL PROCEDURE

Animal Number	*Teeth*	*Operative Technique*	*In Vivo*	*Post Mortem*
Dog 1	7 premolars used	Depth of sulcus measured. Mylar film introduced. Flaps raised and sutured.	Sacrificed ½ hour after operation	
Dog 2	4 premolars used	Flaps raised. Cavities prepared. Plastic fillings placed. Flaps sutured.	Sacrificed 3 weeks after operation	
Dog 3	11 teeth used Incisors and premolars	Depth of sulcus measured. Flaps raised. Flaps sutured. Mylar strips introduced.	Sacrificed 1 hour after operation	
Dog 4	5 teeth used premolars	Flaps raised. Forced from teeth. Replaced. Flaps sutured.	Sacrificed 3 weeks after operation	
Dog 5	17 teeth used Incis. canines premolars molars	Depth of sulcus measured. Mylar film introduced. Flaps raised. Instruments introduced into sulcus.		1 hour after death
Dog 6	8 teeth used premolars	Depth of sulcus measured. Mylar introduced. Flaps raised and sutured.		1 hour after death
Monkey 1	12 teeth used Incisors, premolars and molars	Flaps raised. Mylar introduced. Gingivectomy in premolar and molar area	Sacrificed 3 weeks after operation	
Monkey 2	Same	Same	Sacrificed 6 months after operation	
Monkey 3	Same	Same	Sacrificed 3 months after operation	
Monkey 4	Same	Same	Sacrificed 3 days after operation	
	All specimens were fixed in 10% formalin			

was felt that seemed to be the bottom of the pocket. Using some force, the steel blade could be pushed deeper to a depth of 1.5 mm. A further introduction was prevented by a bending of the blade. To assure a broader separation of gingiva from the tooth, the blade was then moved along the tooth surface mesially and distally.



A. B.

Fig. 5. Dog 5-3. Right lower second premolar. Steel blade was inserted into sulcus with force and moved forward and back. Blade was withdrawn and tooth fixed in 10% formalin solution. A. General view showing gingival margin. A space is visible between enamel surface and the epithelium at X. B. Higher magnification of area between the two arrows in Figure 5A. The steel blade had penetrated to point X, between layers of the epithelium.

After withdrawal of the blade, the specimen was fixed in 10% formalin.

A study of the sections through this tooth (Fig. 5, A & B), reveals that the blade was inserted at first beyond the bottom of the sulcus that was, in all probability, situated at the level of the upper arrow in 5A. From here, the blade progressed in the epithelium between a layer of cells that remained attached to the enamel, and the rest of the attached epithelial cuff. The penetration, with or without force, is located in the same plane. The most important finding is the clear evidence of an injury of the epithelium by the insertion of the steel blade.

All experiments conducted in this manner showed the same epithelial injuries. There was never a separation of the intact epithelial cuff from the tooth surface. Even application of considerable force did not permit the introduction of the blade to the cemento-enamel junction.

II. *Flap and Reposition*

On the same dog used in the first experiment, a gingival flap was outlined by two vertical incisions on the distal half of the

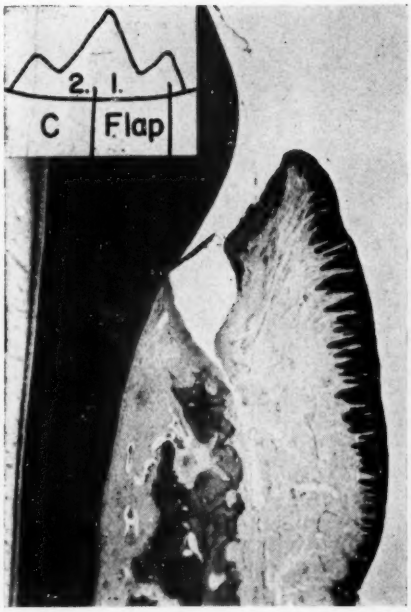


Fig. 6. Dog 5-6. Right lower first molar. Flap prepared on distal half of tooth (see inserted diagram—area 1). Flap was first separated from tooth, then replaced to original position, sutured and jaw fixed in 10% formalin after this operation. The tear in the epithelium as well as the connective tissue is clearly visible.

Fig. 7. Higher magnification of Figure 6. The epithelial attachment marked by X remained attached to the surface of the enamel, while the connective tissue is torn from the epithelium.

right lower first molar. The flap was pulled away from the tooth to the crest of the alveolar bone, then laid back and the specimen fixed in 10% formalin.

The microscopic examination (Figs. 6 & 7) showed that the flap was separated from the enamel for some depth below the bottom of the sulcus. However, at a considerable distance from the cemento-enamel junction, a tear is visible through the epithelium into the connective tissue. From here on, the entire epithelium (X in Fig. 7) remained attached to the tooth. At the cemento-enamel junction, the tear follows the alveolar crest fibers obliquely to the alveolar bone (Fig. 6).

Repeated experiments of the same type, unmistakably showed injuries to the epithelial attachment and the connective tissue. Never could a picture be obtained that was comparable or identical to the normal appearance of the epithelio-enamel relationship pictured in Figure 8. (Control of Figs. 6 & 7).

Separation and replacement of a flap in the living animal and histologic examination after one-half hour survival time, showed essentially the same injuries (Figs. 9, 10-A & B). For some distance, the tear is in the epithelium of the attached cuff, but then reaches through the epithelium into the connective tissue where it can be followed to the alveolar crest.

III. *Insertion of Celluloid and "Mylar" Strips*

If a strip of celluloid is used instead of a steel blade and inserted into the gingival sulcus with great force, it does not separate the epithelium from the tooth surface, but

Fig. 8. The mesial half of the tooth was used as the control. (Diagram area 2, in Figure 6) The gingival margin and epithelial attachment showing no signs of injury. A steel blade could probably be inserted into the sulcus without any appreciable force, to the point at X, but beyond that, force will have to be used.

penetrates into the epithelium. Part of the epithelial cuff remains in its close relation to the enamel surface (Fig. 11).

Following a suggestion made by H. A. Zander,[29] a strip of a thin plastic* was inserted into the gingival crevice. This film is not destroyed during preparation of the specimen (Fig. 12). However, it is not always possible to prevent its loss. But even when lost, its position can be detected in the section because it leaves (Fig. 13) a clear and unmistakable imprint.

If the depth of a sulcus was measured by inserting a steel blade until resistance was felt and if then a mylar strip was introduced, it could never be pushed beyond the measured depth. If more force was applied the strip buckled and the gingival

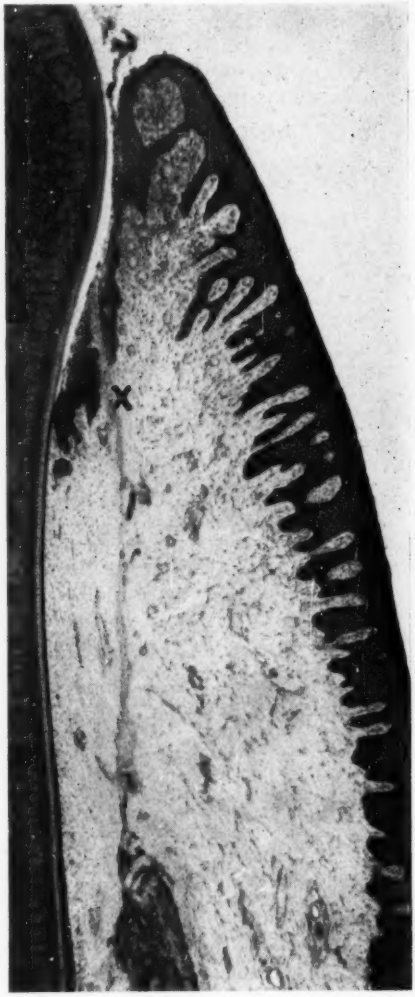
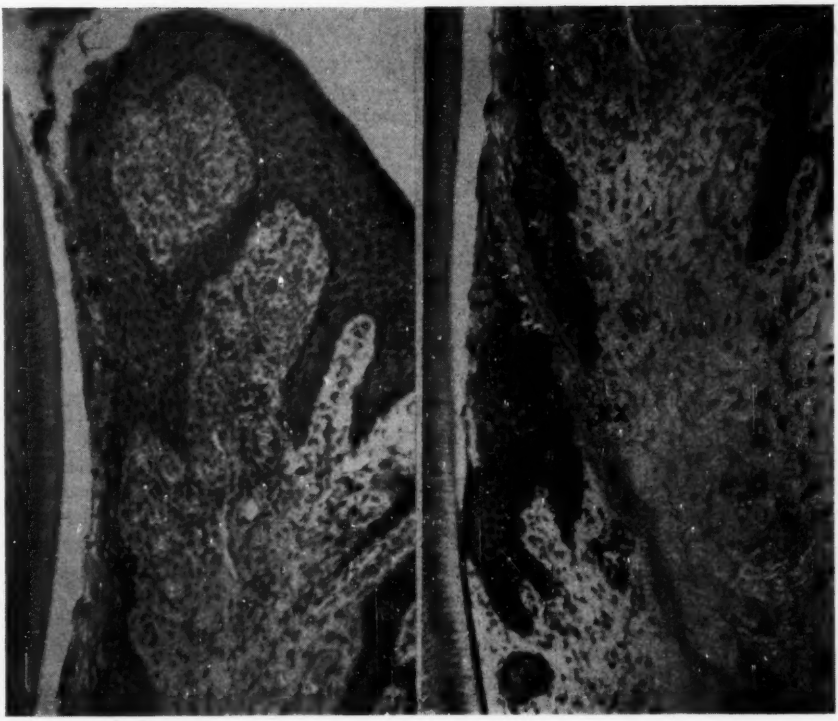


Fig. 9. Dog 1-1. Right lower second premolar. Depth of sulcus measured 0.5 mm. A flap was raised. In the attempt to separate the epithelium, a tear occurred within the epithelium through the connective tissue to crest of the alveolar bone. Flap was replaced. Dog sacrificed ½ hour after experiment was started and specimen fixed in formalin. The tear penetrating through the epithelium and connective tissue is visible as a necrotic tissue line, marked by X.

margin wrinkled. Histologic examination of a tooth thus treated (Figs. 14 & 15) in which a considerable amount of enamel matrix was retained, shows that the steel blade and the mylar strip had been intro-

*A polyester film marketed by Du Pont under the trade name, "Mylar." We are indebted to Dr. Zander for furnishing this material.



A. B.

Fig. 10. Higher magnification of two different areas of Figure 9. In the attempt to separate the flap from the tooth surface, the tear occurred in the epithelium (A) marked by X, and extended into the connective tissue, B. The tear is visible as a necrotic tissue line marked by XX.

duced to the measured depth of one millimeter. But even though this penetration was possible without any force, it occurred within the epithelium. A layer of epithelial cells remained clearly attached to the enamel (Fig. 15A). Farther apically, the primary enamel cuticle and the organic connection between epithelial cells and the cuticle and between cuticle and interprismatic substance is in clear evidence (Fig. 15B).

Identical results were obtained in repeated experiments, whether the insertion was undertaken intra vitam or post mortem. The shallow penetration of the mylar strip is especially striking in the tooth in which much of the epithelium still consisted of well differentiated and functioning ameloblasts (Fig. 4).

DISCUSSION

Waerhaug's claim that from the moment a tooth erupts into the oral cavity, the gingival sulcus reaches to the cemento-enamel junction, is invalid. In a great number, possibly for the great majority of erupting teeth (especially of dogs) that have already been exposed to the oral cavity, enamel formation and/or maturation is still not completed in the cervical areas of the crown (Fig. 4). In this region, fully differentiated and functioning ameloblasts can still be observed. There cannot be any doubt that ameloblasts and their

Fig. 11. Dog 1-4. Right upper first premolar. Original sulcus, 1.5 mm. in depth. Celluloid film was forcibly inserted into the sulcus. Observe that the tear occurred within the epithelial attachment, leaving epithelial cells still attached to the enamel.

rods form an organic unit as long as amelogenesis continues. The practical implications of this simple fact which was neglected by Waerhaug, are obvious. Any interference with the primary connection between ameloblasts and developing enamel must lead to an interference with the last stages of amelogenesis.

The question of an attachment of epithelium to enamel should logically be restricted to the period after amelogenesis has come to a halt. Here, the assertions of Waerhaug are clearly contradicted by the experiments and findings described above.

In attempting to detach a gingival flap from the enamel surface in vivo or immediately after sacrificing an animal, we never were successful in separating the epithelium cleanly from the enamel as claimed by Waerhaug. Sometimes, such a separation may occur for a short distance apical to the bottom of the sulcus. However, farther apically, the force exerted causes tears within the attached epithelium so that the innermost cells remain attached to the enamel. Still farther apically, the tear very often reaches through the entire thickness of enamel epithelium into the connective tissue, and if sufficient force has been applied, to the crest of the alveolar bone.

The signs of such a forceful reflection of a gingival flap were observed in all our specimens, and remain clearly visible, even after careful replacement and suturing the flap to its original position. The latter finding clearly contradicts the opposite claim of Waerhaug, who attempts to show that histologically there is no difference between the attached and replaced gingiva. This striking contrast between Waerhaug's and our findings, allows for only one explanation—namely, that by some mistake, Waerhaug's sections were made from a tooth or an area of a tooth that had not

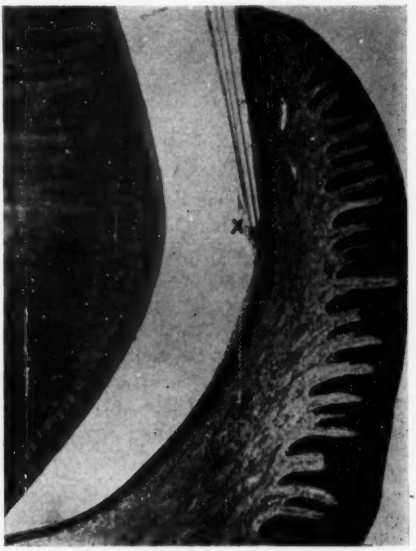


Fig. 12. Dog 5-10. Lower left fourth premolar. Mylar strip in sulcus indicating its depth. Depth of sulcus, 1 mm. from gingival margin to bottom (X).

been subjected to the experimental reflection of the gingival flap.

Our experience that the tear, in reflection of the gingiva, occurs *within the epithelium* is in full accord with observations of Kronfeld,[11] Orban and Mueller,[5] Macapanpan[28] and the latest observations of Ussing.[20] Ussing, working on tooth germs, states that "Histologically, the cuticular isolates appear to consist of a continuous membrane, the enamel cuticle, with attached epithelial cells, which in sections are identified as cells of the enamel epithelium. The greater part of this epithelium is found attached to the tooth surface indicating that the separation observed clinically takes place between the enamel epithelium and the connective tissue of the sac."

Our observations are also in accord with Toller's experiments,[16,17,18] in which he tried to tear the epithelial attachment away from the surface of the enamel under a dissecting microscope. He states, "At levels near the crest of the gingiva, the plane of separation is relatively close to, or at, the enamel surface. At levels nearer the cemento-enamel junction, the separation is within the epithelium of the attachment portion . . . ." Toller even observed tears continuing through the epithelium into the connective tissue (see our Figs. 6, 7, 9 & 10) when attempting to separate a gingival flap from the surface of the tooth.

A second set of Waerhaug's experiments consisted of the introduction of a thin steel

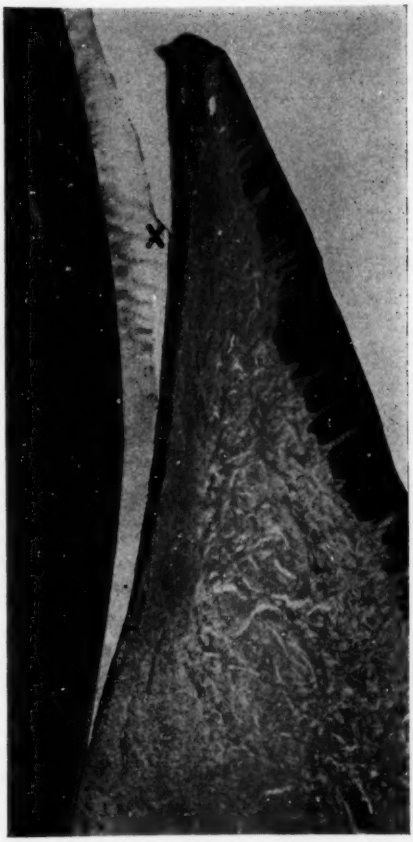


Fig. 14. Dog 5-1. Right lower third incisor. Depth of sulcus, 1 mm. Bottom of sulcus indicated at X. Mylar strip was inserted to this point

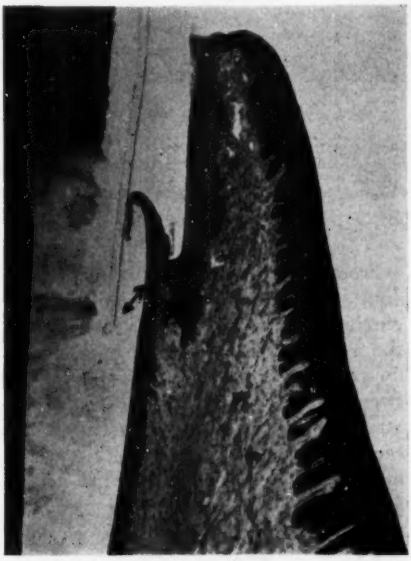
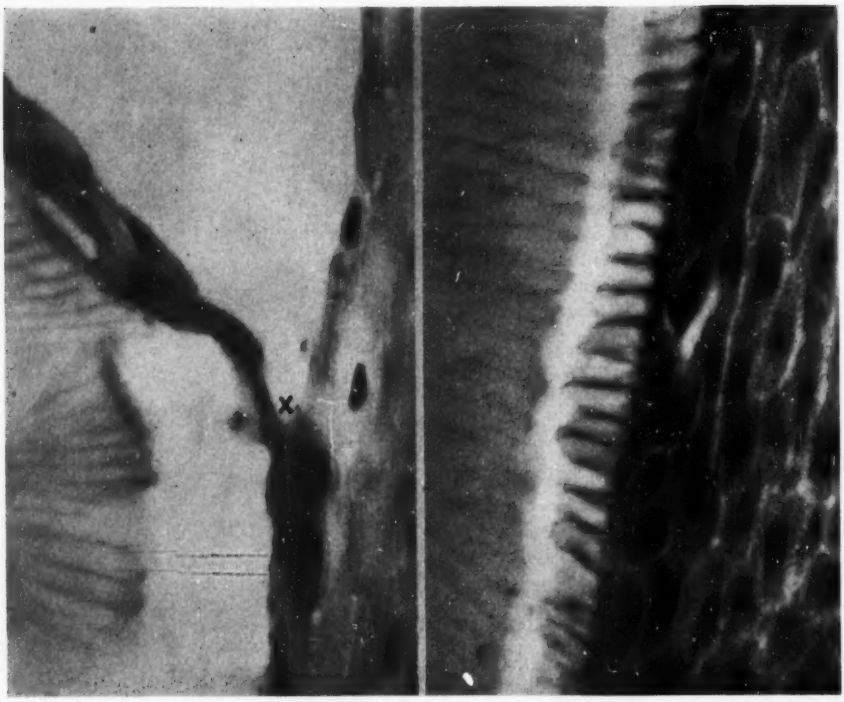


Fig. 13. Dog 5-3. Right lower canine. Depth of sulcus measured 1½ mm. Mylar was inserted into the sulcus to a point where resistance was felt. During preparation of the specimen, the gingiva became loose and the mylar strip was displaced. However, the former location of the mylar strip is unmistakable, as indicated by the shape of the space which it formerly occupied (indicated by arrow.)



A. B.

Fig. 15. Higher magnification of two areas from specimen in Figure 14. A. Bottom of sulcus, X. Mylar was inserted to this point. Epithelial cells are attached to the organic matrix of the enamel. Separation occurred within the epithelial layer. B. In an area about 1.5 mm. apical to the bottom of the sulcus, enamel matrix has been retained with an obvious organic attachment of enamel epithelium to enamel matrix.

blade into the gingival sulcus. He states that a thin strip of steel glides, without application of considerable force, within the sulcus,—that is, between epithelium and enamel surface, to the cemento-enamel junction. Here it is stopped by the attachment of gingival fibers to the cementum. All our attempts to duplicate Waerhaug's findings by using strips of steel, mylar plastic film or celluloid, were unsuccessful. In all cases, these strips penetrated only to, or slightly beyond that point where the bottom of the sulcus could be identified. Apically, the strips always entered the epithelium itself, leaving a layer of cells attached to the enamel or even entered into the connective tissue through the epithelium.

From all these observations, we have to draw the inevitable conclusion that the gingival epithelium is, in the true sense of the word, attached to the enamel surface. Of course, the question remains to be answered as to the mode or mechanics of this attachment. Two alternatives have to be considered: 1. A persistence of the primary union between inner enamel epithelium and enamel rods after completion of amelogenesis, or 2. the establishment of a secondary attachment after this time by the formation of a cementing substance. It should be remembered that Weski[30] claimed that Gottlieb's secondary cuticle had to be interpreted as such a cementing substance.

In connection with the solution of this

as yet unanswered problem of the mode of attachment, the question also has to be considered of how firm or weak the epithelial attachment might be. It certainly is of some firmness because it is evidently stronger than the intracellular connections of the cells of the reduced enamel epithelium and the connections between epithelium and underlying connective tissue.

It is here suggested that the problem of the attachment of epithelium to the tooth surface is not to be discussed as an isolated problem but only as a part of the *dento-gingival junction.* In the gingiva itself, as in skin and oral mucosa generally, the connective tissue of the lamina proporia and the epithelium divide the labor of forming a protective covering of the underlying tissues. Though these functions overlap, it still can be said that mechanical resistance is mainly served by the connective tissue, while the biological security of the underlying tissues (e.g., against chemical or bacterial irritation) is served by the epithelium. In the dento-gingival junction, we see a similar division of functional properties. While the connective tissue of the gingiva, both the fibers attached in cementum and bone, and the fibers surrounding the tooth, guarantee the firmness of the junction between soft and hard tissues, the biologically significant, even indispensable sealing of this point of low resistance is completed by the true attachment of gingival epithelium to the surface of the tooth.

In order to avoid a misinterpretation of this statement, it should be repeated, that the two functions, mechanical and biological, are, of course, present in both tissues, but the emphasis in the connective tissue is on the resistance of mechanical forces, while the emphasis in epithelium is resistance to bacterial and chemical injuries.

The term, epithelial cuff in describing the epithelium that is related to the surface of the tooth, is quite acceptable and indeed, has been used all the time in a purely descriptive sense. It should not imply a lack or a presence of a true attachment of the epithelium to the tooth. It is true that the use of the term "epithelial attachment" for the attached epithelium is linguistically improper. The term, *attached epithelial cuff* could be used instead.

CONCLUSIONS

1. In young dogs it was impossible to detach the gingiva from the enamel of the tooth without causing tears in the epithelium or in the connective tissue.

2. It was also impossible to insert a thin strip of either steel, mylar or celluloid into the alleged space between epithelium and enamel. Instead, such a strip will also cause tears in the epithelium or through the epithelium into the connective tissue.

3. In all these experiments, the injuries caused by the manipulation are clearly and unmistakably. visible in carefully prepared sections.

4. Epithelium is not made adherent to the tooth surface by formalin fixation.

5. The gingival epithelium is in the true sense of the word, attached to the enamel surface.

6. The attachment of the epithelium to the tooth should be considered as part of the dento-gingival junction.

7. It is suggested that the term, "attached epithelial cuff" should be used instead of the term epithelial attachment.

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An Analysis of Physiologic Rest Position, Centric Relation, Centric Occlusion

by PAUL N. BAER, D.D.S., *Los Angeles, California*

THERE has arisen a great deal of confusion in regard to three commonly used terms in dentistry, namely, physiologic rest position, centric relation, and centric occlusion. This confusion may have arisen because the words, "centric," or, "relation," do not describe a physiologic function. Furthermore, a definition of these words do not describe the meaning for which they were intended. This is a real weakness in the use of any terms.

The clinician usually means by physiologic rest position, a position of the mandible when the teeth are apart, and the mandible is suspended, reflexly, by an equilibrium of the tonus of the muscles which tend to elevate or depress the mandible. Thompson[1,2] and Moyers[3] both believe that this position is established early in life, and is unrelated to the presence or absence of teeth. Thompson,[1,2] in his early writings, thought that the physiologic rest position remained constant throughout life. He has since changed his concept of the immutability of the rest position. In a recent article,[4] he now states that variations in the rest position occur and, " . . . are related to variations in the tonicity of the muscular tone such as (1) hypotonicity of generally diminished muscular tone, and (2) hypertonicity, which in the extreme is muscle trismus."

Thompson is, therefore, now in agreement with Moyers[3] who believes that physiologic rest position is a "relatively stable" position, rather than an absolutely stable one.

How is the physiologic rest position supposed to differ from centric relation? The older, more classic way of defining centric relation, is to say that the mandible is in centric relation when both condyles are in the most retruded position in the glenoid fossa from which lateral and protrusive movements can be made at a given degree of jaw separation. By defining it in this way, the clinician meant centric relation to be a much more definite position than physiologic rest. Sears[4] differentiates between centric relation and physiologic rest as follows: Before speech begins the mandible hangs loosely in what is termed the rest position. Just as centric relation is a point of reference for the discussion of mechanical problems, so is the rest position a point of reference for phonetic problems. The rest position is not so definite a one as centric relation. It is merely an habitual position of the mandible when not in use.

We can now see the confusion in terminology starting to develop. According to this concept, physiologic rest position is no longer the fixed position, but rather centric relation is supposed to be the definite one. The idea of a definite or fixed centric relation, however, is just as objectionable as is a fixed physiologic rest position. As Moyers[3] states, "The concept of a static centric relation is not only misleading, it is contrary to what is known of neuromuscular physiology." Moyers defines centric relation as, "The position of the mandible as determined by the neuro-muscular reflex first learned for controlling the mandibular position when the primary teeth were in occlusion."

Fox,[6] on the other hand, defines centric relation as a, "Practiced, learned, neuromuscular pattern and a position of mandible to maxilla based on a neuromuscular pathway ranging from physiologic rest position to terminal tooth contact, when a posterior, unstrained, hingelike movement of the mandible is effected. Centric relation would, therefore, vary according to the (1) existing tonus of the muscles of

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the articulation and (2) the vertical dimension at which the positional relationship is established."

The conclusion one can draw from both of these definitions is that centric relation is a pathway, rather than a fixed position.

Moyers further differentiates centric relation from physiologic rest position of the mandible, by saying that centric relation is a "learned" neuromuscular reflex, while physiologic rest position is an "unlearned" position, and is, in fact a postural reflex. He then draws the following conclusions: "The latter (physiologic rest position) is a postural reflex and is not effected by stimuli from the periodontal receptors as is centric relation."[2]

Since in so-called, "physiologic rest position" the muscles are in fact, actually in a state of mild muscular contraction, one would have to agree with Moyers, that physiologic rest is really a postural reflex. Being a postural reflex, it is controlled mostly by the "stretch reflex." The "stretch reflex" works by stimulating a muscle to contract when the proprioceptors located in the muscle are stretched. However, it is extremely important to remember that this "stretch reflex" can itself be controlled by the higher centers in the spinal cord and brain, which can either reinforce or inhibit it. Thus, the postural position of the jaw, more commonly spoken of in dentistry as physiologic rest, can be expected to vary in any condition which might change the tonus of muscle, such as, fatigue, and state of mental and physical health. However, one must not forget, that stimulation of proprioceptors locally can also effect muscle tonus. Therefore, contrary to the opinion of Moyers, stimulation of the proprioceptors from nerve endings in the periodontal membrane and even the gingivae, can play an important role in establishing the physiologic rest position of the mandible. As Atwood[7] states, ". . . local proprioception from nerve endings in the periodontal membrane must play a large part in the natural inhibition of the jaw-closing muscles—another of the body's self defense mechanisms, for if there were no such reflex inhibitions of the jaw-closing muscles, traumatic occlusion would be universal, and bruxism would lose its psychiatric connotion. When the teeth are extracted, the rest position of the mandible may tend to close to a new equilibrium because of the loss of the normal inhibition of the tone of the closing muscles."

It becomes apparent, therefore, that neither physiologic rest, or centric relation are fixed positions, but that both can vary as to whether we are standing erect or bending over, whether teeth are present, absent, or erupting, as well as in various states of mental and physical health. It now becomes more obvious that centric relation and physiologic rest are really one and the same, and that all these years clinicians have been attempting to differentiate between two conditions which are really identical. They are simply a postural jaw position. It seems, therefore, that the term centric relation could be omitted. The postural jaw position can be defined as the position the jaw assumes when the teeth are apart and the mandibular position is maintained by the "stretch-reflex." It is variable to the extent that the stretch reflex can be modified through local proprioception and the higher centers. Its main clinical significance is found in various phases of mouth rehabilitation where it becomes important that in a postural jaw position, a proper free-way space should exist. It may also play a role in disturbances of the temporomandibular joint.

With this definition established, centric occlusion can now be differentiated rather easily from the postural jaw position, by defining it in its true physiologic terms. Centric occlusion is the contact position of the teeth, when the mandible is brought to its most retruded position by a physiologic act, which most often is the act of swallowing.

SUMMARY

Physiologic rest position and centric relation apparently are one and the same

position, and the latter term could be discarded.

The mandible can now be said to have but two positions which have clinical significance in the field of dentistry. One is a contact position of teeth, which is centric occlusion, and the other, is the postural position of the jaw.

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Quantitative Evaluation of the Effect of Toothbrushing and Toothpicking

by TORMOD MORCH, D.D.S., PH.D.,* AND JENS WAERHAUG, D.D.S., PH.D.,* *Oslo, Norway*

IN clinical dentistry oral hygiene has been considered one of the most valuable adjuncts in the management of periodontal disease. Recent histologic findings have demonstrated the beneficial effect of the removal of subgingival calculus for the conditions of the gingiva.[1] In a study on the presence or absence of bacteria in gingival pockets it was observed that bacteria as a rule were found only in pockets where calculus or soft deposits were retained on the tooth surface below the gingival margin, and, that the bacteria disappeared from the pockets when the deposits were removed.[2] These findings suggest that bacteria, retained in contact with the gingiva, are the most important causative agents in the establishment of gingival inflammation and subsequent destruction of the supporting tissues.

The bacterial flora of dental plaque is a mixture of a vast number of species and there is reason to believe that dental plaque is an important source of the bacteria in the saliva. Some of the bacteria in the saliva produce enzymes which break down saccharides with the production of acids. *Stephan & Hemmens*[3] state that the masses of microorganisms growing on the teeth produce a rapid drop in pH when carbohydrates are ingested, and that this drop is related to the amount of bacteria. It is known that the acid producing bacteria always represent a part of the whole flora. Therefore, a measurement of the acid production allows a quantitative evaluation of the bacterial status of the oral cavity.

During the brushing the bacterial plaque is transferred from the teeth to the saliva and the same happens during toothpicking. If these procedures are carried out without the use of tooth paste, the saliva will contain all, or most of the bacteria from the dental plaque, and by collecting this saliva and adding sucrose, its acid producing capacity can be measured.

The purpose of this study is to evaluate the ability of breaking down sucrose by saliva samples collected: (1) before

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cleansing of the teeth, (2) during toothbrushing, (3) during toothpicking and (4) after cleansing of the teeth. The data from such an experiment may throw some light upon: (a) the relative amount of bacterial plaque removed by the toothbrush and the toothpick, and (b) the bacterial status before and after oral hygiene. By varying the time allowed to elapse after the last cleansing of the teeth, one can get an idea about the speed with which the bacterial plaque is formed, and how often it should be removed in order to obtain the optimal conditions for a healthy state of the gingiva.

MATERIAL AND METHODS

For the experiments were selected 4 healthy adults, (1: age 45, 2: age 38, 3: age 27 and 4: age 48) two of them using toothpicks habitually, and two of them being instructed to do so at the beginning of the experiment. After a thorough cleansing by toothbrush and toothpicks, the experimental persons refrained from any kind of oral hygiene for observation periods varying from 2 hours to 6 days. Saliva samples of 6 ml. were collected successively in the following way:

Sample I: *Before oral hygiene* without any special stimulation.

Sample II: *During brushing.* The teeth were brushed continuously during the collection of the samples, and the saliva was sucked out of the toothbrush.

Sample III: *During toothpicking.* After a thorough rinsing with distilled water saliva samples were collected during continuous toothpicking; teeth were sucked intermittently.

Sample IV: *After oral hygiene.* Again the mouth was rinsed with distilled water, and the samples were collected, some with and some without paraffin stimulation.

The samples were kept at 37°C throughout the experiment as far as this was possible. Each sample was stirred in a beaker till all the excess of carbon dioxide was removed, i.e. till the pH readings showed constant values. By this procedure it was prevented that escape of carbon dioxide caused a rise in pH during the experiments. Then 5 ml. of each sample were transferred to a 10 ml. measuring-glass and 1 ml. of a

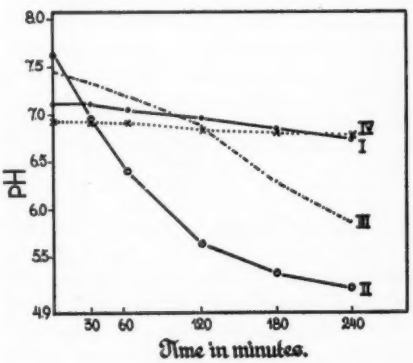

Fig. 1. Line graph illustrating drop in pH in relation to fermentation time of saliva samples from individual 4, after 16 hours without oral hygiene. Samples collected (I) before cleansing, (II) during toothbrushing, (III) during toothpicking, and (IV) after cleansing.

50% sucrose solution was added. The amount of bacterial matter present in each sample was determined by its capability of producing acids through fermentation of the sucrose. The acid production was estimated by pH readings with a glass electrode.* The first reading was made immediately after the addition of sucrose (time: 0) and the following ones after ½, 1, 2, 3 and 4 hours.

RESULTS

The results are given in Tables I to V

*The titration method, which would have given more absolute values, was not used in this pilot study.

and the graphs made from these tabulations are shown in Figures 1 to 5.

Table I and Figure 1 demonstrate the drop in pH in relation to the fermentation time in the four separate saliva samples from one experiment person, (4) after 16 hours without oral hygiene. The corresponding drop after 6 days is shown in Table II and Figure 2. It is apparent that the greatest drop in pH takes place in the toothbrushing samples, somewhat less in the toothpicking sample and least in the sample collected after tooth cleansing. The drop in pH also starts more quickly in the toothbrushing sample than in any of the other samples. Tables I and II and Figures 1 and 2 are representative of all the experiments in which the individual had refrained from oral hygiene for at least 12 hours.

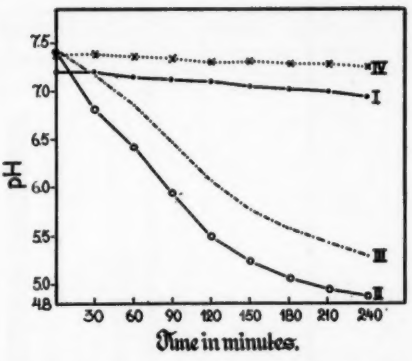

Fig. 2. Line graph illustrating drop in pH in relation to fermentation time of saliva samples from individual 1, after 6 days without oral hygiene. Samples collected (I) before cleansing, (II) during toothbrushing, (III) during toothpicking, and (IV) after cleansing.

TABLE 1

Drop in pH in relation to fermentation time in saliva samples collected after 16 hours without tooth cleansing. Individual 4.

Time, min.	*Before cleansing*	*During tooth-brushing*	*During tooth-picking*	*After cleansing*
0	7.11	7.63	7.47	6.94
30	7.11	6.95	7.34	6.94
60	7.06	6.39	7.20	6.91
120	6.97	5.62	6.88	6.84
180	6.86	5.31	6.30	6.81
240	6.73	5.16	5.87	6.76

The variation in pH in saliva samples collected from the four experimental persons after 2 days without oral hygiene is illustrated in Table III and Figure 3. As was the case in the foregoing illustrations, the drop in pH for each individual was greater in the toothbrushing samples than in the toothpicking samples. The samples collected after cleansing showed a somewhat slighter drop than those collected before. A certain individual variation can be observed.

The variation in pH in relation to the length of time without oral hygiene is illustrated in the Tables IV and V and the Figures 4 and 5. The highest values in each instance were in both series found in the toothbrushing samples. The fall in pH increased conspicuously with the length of time without oral hygiene in both the toothbrushing and the toothpicking samples, but the tendency was most pronounced in the latter.

DISCUSSION

The most remarkable finding in this series is the rapid drop in pH in the toothbrushing and toothpicking samples as compared with those collected before and after

TABLE 2

Drop in pH in relation to fermentation time of saliva samples collected after 6 days without tooth cleansing. Individual 1.

Time, min.	*Before cleansing*	*During tooth-brushing*	*During tooth-picking*	*After cleansing*
0	7.15	7.35	7.38	7.32
30	7.15	6.78	7.12	7.32
60	7.10	6.39	6.82	7.30
90	7.08	5.92	6.45	7.28
120	7.06	5.49	6.05	7.25
150	7.01	5.24	5.76	7.26
180	6.98	5.06	5.58	7.23
210	6.96	4.95	5.43	7.23
240	6.90	4.88	5.30	7.21

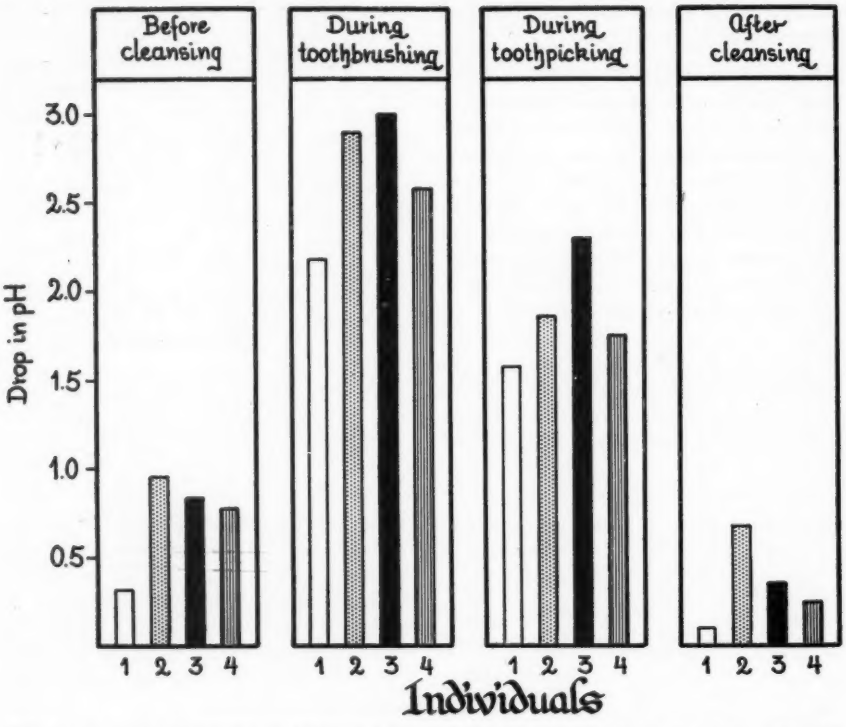

Fig. 3. Bar graph illustrating variation of drop in pH of saliva samples from all four experimental persons, after 2 days without oral hygiene. Samples collected under different conditions specified in Figure.

this procedure. Thus it is obvious that the two samples collected during cleansing of the teeth contained more bacteria capable of producing acids than those collected before and after. There should be little doubt that the majority of these acid producing bacteria combined with a vast number of others, have been retained on the teeth in the form of plaque; and some of this bacterial plaque has been lying in direct contact with the gingivae as demonstrated in Figure 6. (In this case the bacteria caused a severe inflammation in the gingivae with deepening of the pocket as a result). Consequently, this method allows a quantitative evaluation of the effect of toothbrushing and toothpicking for the prevention of gingival inflammation and periodontal disease. The more rapid the drop in pH is, the more bacteria have been removed from contact with the teeth and the gingivae.

This series demonstrates also that brushing alone does not remove all the bacteria from the teeth, as a considerable drop in pH was observed in the samples collected

TABLE 3

*Drop in pH after 2 days without tooth cleansing. A comparison of 4 individuals. *Collected during paraffin chewing*

Individual	*Before cleansing*	*During tooth-brushing*	*During tooth-picking*	*After cleansing*
1	0.32	2.19	1.58	0.10
2	0.95	2.91	1.87	0.68*
3	0.83	3.00	2.30	0.36*
4	0.79	2.58	1.75	0.25*

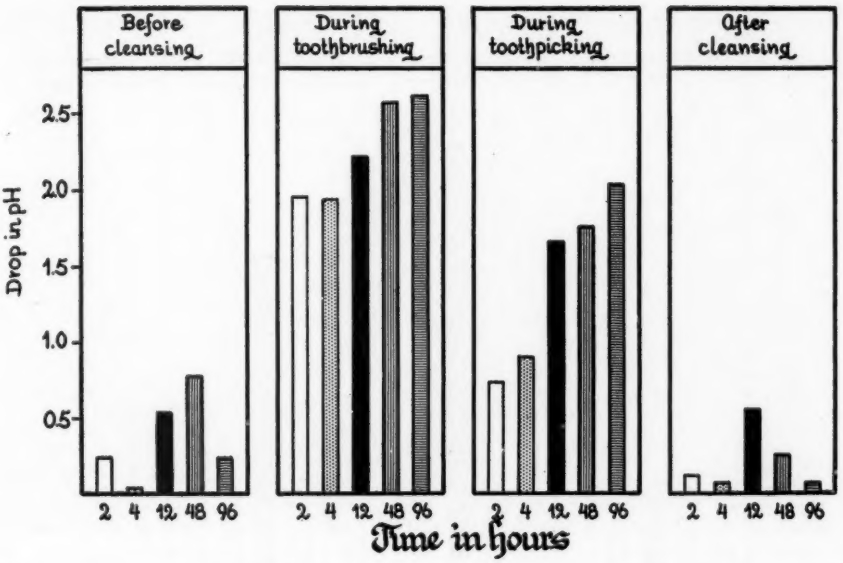

Fig. 4. Bar graph illustrating variation of drop in pH in relation to length of time since last oral hygiene as manifested in individual 4.

during toothpicking. This is easy to explain when looking at the photomicrograph in Figure 6 which is from the interproximal space between the abutment tooth and a pontic. A brush can never reach such areas, whereas a toothpick can do it readily; and it must have been the bacterial plaque located as illustrated here, that mainly caused the drop in pH in the toothpicking samples. For clinical periodontia this demonstrates that toothbrushing alone is not able to remove the bacterial plaque in the interproximal spaces.

TABLE 4

Drop in pH in relation to length of time without tooth cleansing. Individual 4.

Hours	*Before cleansing*	*During brushing*	*During picking*	*After cleansing*
2	0.24	1.97	0.75	0.12
4	0.02	1.95	0.89	0.05
12	0.53	2.22	1.66	0.57
48	0.79	2.58	1.75	0.25
96	0.24	2.62	2.05	0.06

However, when comparing the drop in pH in the brushing samples with those collected during toothpicking, it is apparent that the majority of the bacteria are removed by means of the brush. Consequently, a considerable effect can be attained by means of the brush, although the toothpick (or dental floss)is essential for full efficiency. To what extent subgingival bacterial deposits, inaccessible to the toothbrush and the toothpick, have influenced the results in this study cannot be ascertained. None of the experimental persons had detectable subgingival calculus. However, in spite of this, uncalcified and invisible plaque may have been present.

When the drop in pH is considered in relation to the length of time since the last oral hygiene, it is obvious that the bacterial accumulations increase with time (Figs. 4 and 5), which should be expected. The accumulation takes place faster on the surfaces reached by the brush than on those reached by the toothpick. The reason for this may be that the deposits on the teeth

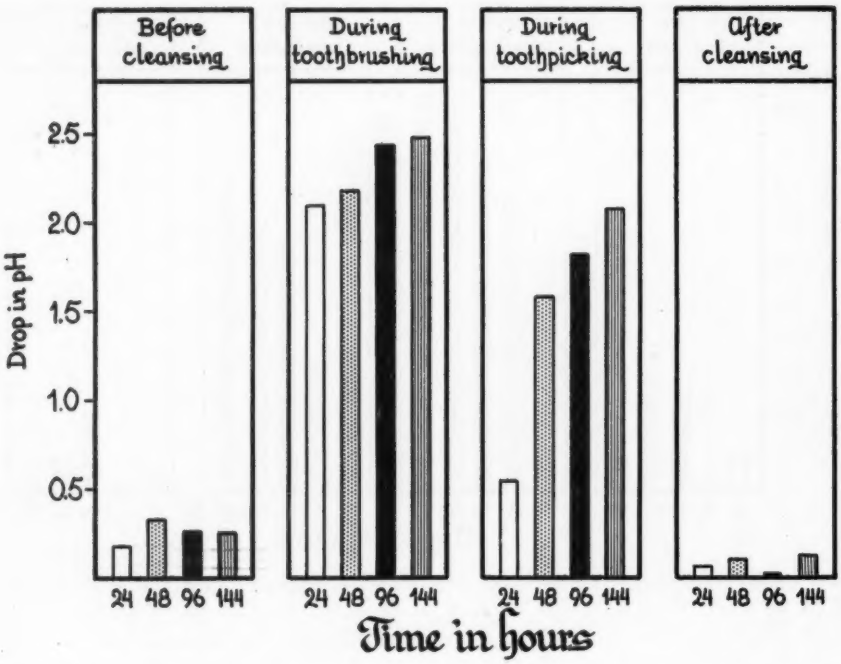

Fig. 5. Bar graph illustrating variation of drop in pH in relation to length of time since last oral hygiene as manifested in individual 1.

are very soft in the beginning and harden with time. Thus, the brush may be able to detach more plaque in the interproximal spaces after a short period than after a long one, when it has got time to harden. However, it was rather unexpected to find a considerable drop in pH in the samples collected after as short periods as 2 to 4 hours (Fig. 4). This rapid accumulation of bacteria was most pronounced in the brushing samples and the explanation may be that the person in question purposely had eaten a meal shortly after the first oral hygiene, and that food remnants were left on the buccal and lingual surfaces more than on the interproximal surfaces. Addition of bacteria from the tongue and throat must be considered also, and these may be more readily brought into the saliva during the brushing. Bacteria, retained in invisible subgingival plaque, may also have played a role, some of them being squeezed out of the pockets during the second cleansing.

Maximum accumulation of bacteria on the surfaces accessible to the brush seems to be reached after 2 days (Fig. 4) to 4 days (Fig. 5) and on the surfaces accessible to the toothpick it is reached somewhat later, after 4 days (Fig. 4) and 6 days (Fig. 5). On the latter surfaces, the peak

TABLE 5

Drop in pH in relation to length of time without tooth cleansing. Individual 1.

Hours	*Before cleansing*	*During tooth-brushing*	*During tooth-picking*	*After cleansing*
24	0.17	2.11	0.54	0.04
48	0.32	2.19	1.58	0.10
96	0.26	2.44	1.82	0.01
144	0.25	2.47	2.08	0.11

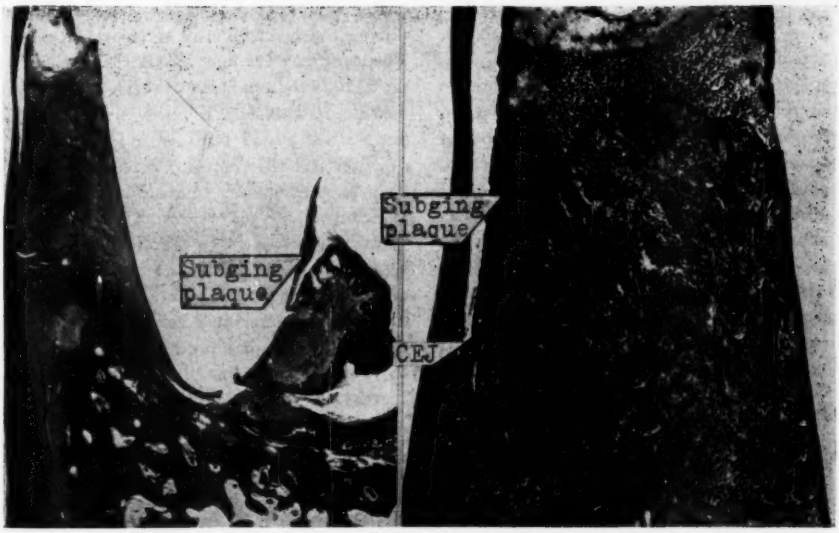

Fig. 6. Photomicrograph from area between abutment tooth and pontic in young healthy monkey. Observation period 329 days. Acrylic root tip dissolved in chloroform after fixation. Soft tissue adjusted to root tip without mentionable signs of irritation. Between pontic and tooth accumulation of bacterial plaque. Subjacent to plaque severe inflammation in connective tissue, and downgrowth of epithelium on abutment tooth.

may be reached even later. This was not determined because of the extreme inconvenience for the experimental persons refraining from oral hygiene for more than 4 to 6 days.

If the assumption is right that elimination of bacterial plaque is important for the prevention and therapy of periodontal disease, this series suggests that the teeth should be brushed two times every day and that the toothpick should be used once. Satisfactory results may be obtained even with the half of this frequency.

In all the samples collected before oral hygiene there was some drop in pH showing that the saliva always contains some acid producing bacteria. If these pH readings are compared with those from the samples collected after the cleansing, it is seen that the drop is smaller in the latter. This clearly demonstrates that the plaque on the teeth is an important source of the salivary bacteria, although not the only one. Other sources may be the tongue,[4] the crypts of the throat and even the nasal cavity. It is probably bacteria from these sources that caused the slight drop in pH in the samples collected immediately after oral hygiene (Figs. 1-5). In the different samples collected before brushing in the same person (Figs. 4 and 5) there was always some variation in the pH reading. The same explanation may be applied for this variation as well. Paraffin stimulation did not seem to influence the acid production mentionably (Table III).

The agreement in the pH readings of the samples collected from the four individuals under different conditions (Fig. 3) justifies the assumption that the fermentation capacity gives reliable values for the evaluation of the relative amounts of bacterial matter in the samples.

It must be assumed that the buffer system of the saliva to some extent neutralizes the acid production in the samples, which means that part of the acid does not effect a drop in pH. Accordingly, no drop in pH

of a sample does not necessarily mean that no acid has been produced. It must also be mentioned that variations in the buffer capacity may have influenced the values of the pH readings. However, these variations are probably small in relation to the buffer capacity itself and especially in relation to the acid production of the saliva sample collected. Therefore, the drop in pH may not have been influenced much by the variations in buffer capacity, and it seems justified to consider the obtained results reliable for a comparison of the variation in pH in saliva samples. It is obvious that this method does not give absolute values.

CONCLUSIONS

1. The efficiency of toothbrushing and toothpicking can be evaluated quantitatively by the fermentation capacity of saliva samples collected during these procedures.

2. The main part of the bacterial plaque on the teeth can be removed by toothbrushing.

3. In areas inaccessible to the toothbrush there will always be some bacterial plaque left, that can only be removed by the toothpick (or dental floss) which demonstrates the need of toothpicks for a satisfactory oral hygiene.

4. The bacterial plaque is quickly renewed after oral hygiene.

5. The amount of bacterial plaque accumulated on the teeth is to a certain extent proportional to the length of time without oral hygiene.

6. The teeth should be brushed two times a day and toothpicks should be used once every day.

SUMMARY

In 4 persons, aged 27 to 48 years, saliva samples (6 ml.) were collected successively before oral hygiene, during toothbrushing, during toothpicking and after oral hygiene. The samples were kept at 37°C and stirred in a beaker until the excess of carbon dioxide had escaped. To 5 ml. of each sample was added 1 ml. of a 50% sucrose solution and the drop in pH was estimated by a glass electrode: immediately after the addition of the sucrose and after ½, 1, 2, 3 and 4 hours. A rapid drop in pH was observed in all the toothbrushing and toothpicking samples demonstrating that acid producing bacteria had been removed from the teeth. The acid producing capacity of the toothbrushing samples was greater than that of the toothpicking samples, indicating that most of the bacteria is removed by the brush. However, the considerable drop in pH in the toothpicking samples shows that some other implement (toothpick or dental floss) is essential for a fully effective oral hygiene. The drop in pH increased with increased time since last oral hygiene, showing that the accumulation of bacterial plaque to a certain extent is proportional to the length of time since oral hygiene.

The findings are discussed in relation to the prevention and therapy of periodontal disease and the conclusion is drawn that for full efficiency the teeth should be brushed two times a day and the toothpick should be used once a day.

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A Mathematical Analysis of the Function of the Fibers of the Periodontal Membrane

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THE manner in which the tooth is supported in its bony socket has received considerable attention. The direction and arrangement of the fibers of the periodontal membrane suggest the simple explanation that the tooth is completely supported by these fibers but certain discrepancies appear with more detailed analysis.

One of the obstacles to accepting the fiber support theory is the waviness of the fibers. Much depends on how much the waviness adds to the fibers' length. If it is enough to permit compression of the membrane before the fibers elongate sufficiently that their total stress is equal to the load, an entirely different mechanism of support comes into play. We investigated this point and offer, in explanation of the waviness, a mechanism in which it would be a mechanical advantage.

In order to simplify the analysis it is confined to the special case of axial forces on the right conical root. This in no way detracts from the validity of the arguments.

TOOTH SUPPORTED BY PRINCIPAL FIBERS

It is a comparatively simple task to set up an equation giving the tension of the fibers in terms of the load or visa versa in symbols. However, to obtain numerical values, important data, the total cross-sectional area of the fibers and their modulus of elasticity, are required and these data are not available.

We can, however, examine some aspects of unit tension T without knowing the modulus of elasticity E.

$$T = \delta E$$

Where δ is the unit strain, i.e. the amount of stretch per unit length.

We assume that the tooth undergoes a given axial displacement. Let l_1 be the straight line distance between the points of attachment of a fiber before displacement and l_2 the distance after the displacement. Now let bl_1 be the length of an unstressed wavy fiber where $b \geqq 1$. The length of the stressed wavy fiber will be l_2 if the displacement of the tooth is sufficient to take the slack out of it. This will be assumed, otherwise the tension would be zero.

Let us write cl_1 for l_2 where $c \geqq b$.

Since $\delta = \dfrac{l_2 - l_1}{l_1}$ for the straight fiber

we write $\delta = \dfrac{cl_1 - bl_1}{bl_1}$ for the wavy fiber

whence $\delta = \dfrac{c - b}{b}$.

To obtain numerical values we assume a tooth with a right circular conical root with a half angle of 14.5°, a periodontal membrane 0.250 mm thick and fibers making an angle of 45° with the horizontal. For an assumed axial displacement of 0.025 mm we find that δ for a fiber with zero waviness ($b = 1$) is 0.040 mm.

The following is the table of values for δ expressed in terms of b, c remaining constant at 1.040.

TABLE I

b.	1.000	1.0025	1.005	1.010
	1.020	1.030	1.040	
δ	.040	.037	.035	.030
	.019	.0097	0	

Thus we see that if the waviness of the fibers adds as much as 4% to their length they will be unstressed for our model until the axial displacement of the tooth exceeds .025 mm. Whether or not the fibers can be stressed depends on how much additional displacement is required and what fraction of the membrane is parenchyma and what capillary blood.

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DISPLACEABLE BLOOD VOLUME

The amount of blood in unit volume of tissue depends on the diameter of the capillaries and their spacing. The diameter of the capillaries in the periodontal membrane is given as approximately .008 mm and examination of the periodontal membrane of a dog showed a spacing about 10 times the diameter of the capillaries.

We may arrive at an approximate value for the capillary volume by assuming that the membrane is divided into cubes with dimensions equal to the size of the capillary mesh n expressed as the number of capillary diameters and we may assume that the axis of a capillary is the line along which 4 such cubes join.

If l is the length of an edge of a cube, i.e. the size of the mesh, $\frac{1}{l^3}$ is the number of such cubes per unit volume, in this case per cubic mm. Since each cube has 12 edges and each edge involves 4 cubes, the total length of a capillary for each cube is $3l$. The volume of blood associated with each cube of the mesh is $3\pi\left(\frac{d}{2}\right)^2 l$ and V_B the total volume of blood per unit cube is $3\pi\left(\frac{d}{2}\right)^2/l^2$

But $l = n \times d$. Therefore

$$V_B = \frac{3\pi}{4n^2}$$

Substituting $n = 10$ gives

$$V_B = 2.35 \times 10^{-2}$$

Thus 2.35% of the total volume of the periodontal membrane is displaceable blood.

DECREASE IN VOLUME OF PERIODONTAL SPACE

A close approximation of the volume of the periodontal space is given by

$$V_P = Ah$$

where A is the area of the root and h is the thickness of the membrane.

In the same way the decrease in volume V_P' due to loading can be written

$$V_P' = u A \sin \alpha$$

where u is the depression of the tooth and α the half angle of the apex. $u \sin \alpha$ is, therefore, the component of the displacement perpendicular to the surface of the root.

The fractional part of the volume of the periodontal space lost by loading is

$$\frac{V_P'}{V_P} = \frac{u \sin \alpha}{h}$$

Substituting the values of u, h and α gives

$$\frac{V_P'}{V_P} = 2.5 \times 10^{-2}$$

Thus the volume of the periodontal space is reduced 2.5% by loading the tooth so as to depress it .025 mm.

We see, therefore, that for our model tooth the volume of displaceable blood is nearly equal to the volume of the periodontal space lost by depressing the tooth .025 mm.

Therefore, it appears (1) that a depression u of .025 mm or .001 inch is approximately the limit to which this particular tooth can be depressed without causing the parenchyma to be compressed and (2) that whether or not the fibers alone can support the tooth depends on their (a) waviness and (b) modulus of elasticity.

From Table I we see that if the fibers had a waviness that added as much as 4% to their length they would be entirely unstressed and all resistance would come from the compressive stress of the membrane. If their waviness added only 2%, δ would be less than half that for a straight fiber and the modulus of elasticity would have to be more than twice as great as for a straight fiber if it is to prevent compression of the membrane.

This argument assumes that the fibers themselves have zero stiffness, which is probably a very close approximation to the fact because of their low elastic moduli and very small moment of inertia.

TOOTH SUPPORTED BY PRESSURE OF THE PERIODONTAL MEMBRANE

If, as appears to be the case, the collagenous fibers alone do not support the tooth, the compression of the parenchyma of the periodontal membrane must supply this support or part of it.

Due to the thinness of the membrane this proposition cannot be treated as an ordinary case involving the moduli of elasticity and shear. This may be made clear by considering first the prism of material in which the dimensions of its cross-section are comparable to its thickness or height.

If such a prism is compressed its thickness is decreased and the dimensions of its cross-section are increased a fraction (Poisson's ratio) of the amount the thickness is decreased.

However, if the thickness is very small in proportion to the area, we are dealing with a membrane. Here a very small decrease in thickness of an incompressible or nearly incompressible substance may result in a great increase in the area of the membrane. This means that part of the membrane may be greatly displaced laterally or tangentally.

For example consider a membrane 0.01 inch thick, 0.3 inch long and of any width, held between two rigid plates and confined on the two sides and one end. If such a membrane is subjected to a force sufficient to reduce its thickness 0.001 inch it will extrude 0.033 inch at the free end so that the tangental displacement of the free end will be 33 times the normal displacement.

The pressure within the membrane which resists the load is made possible by the membrane's resistance to shear (viscosity of a fluid). It is obvious from this and the facts pointed out above, that a thin membrane which is practically incompressible could resist a large load with a very small normal displacement even tho the shear modulus is finite.

Synge[9] applied these principles in a mathematical analysis of tooth support by a membrane, incompressible because of its non-vascular fluid content, and determined the amounts of movement and pressures in various parts of the membranes of model teeth for forces applied at various angles to the tooth axis. In an axially loaded tooth the pressure is a maximum at the apex and atmospheric at the gingival margin. He also determined the centers of rotation under these forces. His calculations satisfactorily explain the tightness of the teeth and give values for the center of rotation that agree well with observed values. The theory also accounts for the loosening of teeth with congestion of the periodontal membrane and the fact that teeth remain depressed or rotated for some time after the sustained pressure of setting an inlay or bridge or of separation.

The incompressible membrane theory has the advantage over the fiber support theory that, after forcing the vascular blood out of the tissue, the resistance builds up with great rapidity with a very small displacement of the tooth as against the displacement required to put the fibers under sufficient tension to resist the load.

Hay[1] also worked out a solution of this problem assigning to the membrane a small compressibility. As the tissue contains much water he used the bulk modulus of water as a measure of compressibility. When applied to similar models Hay's numerical results differed but little from Synge's.

Neither Synge nor Hay assign any function to the collagenous fibers except that Synge says that "it is difficult to see how they alone could account for the tightness possessed by a tooth" and that "it is more probable that their effect on the elastic specification is to render the membrane anisotropic." He does not, however, take such anisotrophy into consideration.

COMBINED FIBER AND INCOMPRESSIBLE MEMBRANE THEORY OF TOOTH SUPPORT

The number and arrangement of the collagenous fibers suggest, in spite of the apparent inadequacy of the all-fiber support theory, that the fibers do play some part other than to render the membrane anisotropic. This suggestion is stronger in view of the high pressures in the membrane under occlusal loads within the upper limits of those actually measured as determined by Synge's equations.

Schour[7] says "A short distance from the surface of the root they (the bundles of fibers) break up into smaller bundles which anastomose and interlace, passing around the blood vessels and fibers in their course and again

uniting into large bundles for attachment at their other extremity."

This suggests that the fibers form a sort of net-work which serves as a hammock or sling to act as a support or resistance against a displacing force. If the fibers were stretched merely as a result of displacement of the tooth there would be no apparent advantage in this arrangement.

We, therefore, investigated the possibility that the pressure (as calculated by Synge's theory of the incompressible membrane) causes a tangental displacement of the membrane, which is opposed by the network of collagenous fibers and that the resulting tension of the fibers assists in the support of the tooth. In other words, the fibers assume the form of a catenary and resist tangental forces in the same manner as the cables of a suspension bridge resist vertical forces.

It is necessary, therefore, to consider the tangental displacement of a point in the membrane in relation to other points at varying distances measured in the direction from root to bone or the reverse.

Synge[10] points out that the principal parts of the tangental components of the displacement in the membrane are proportional to $x(x - b)$ where x is measured in the direction of the thickness of the membrane. This gives a parabolic curve for tangental displacement plotted againt x, the displacement being zero at the bone and the root of the tooth and a maximum in the center of the membrane.

This is the form that a catenary with a load uniformly distributed horizontally* would assume. The pattern of tangental displacements across the membrane, therefore, "fits" the curve the fibers would assume under the type of load applied.

The fact that the tangental displacement in the membrane may be a number of times as great as the normal displacement would account for the waviness of the fibers, for, as will be shown, if there is not some slack or sag in the fibers the tension would become too great.

In our calculations we used the same right circular cone-shaped tooth with a half angle of 14.5°, a root length of 12.4 mm with a membrane whose thickness is 0.25 mm (0.01 inch) and whose fibers run at 45° to the horizontal. We considered that the tooth is displaced axially 0.030 mm, 0.025 mm of which displaces the capillary blood before compressing the membrane.

Letting X equal the total resistance of the tooth to the load, X′ the part of the resistance due to pressure in terms of displacement as calculated by Synge and T_v the vertical component of the total stress of the fibers

$$X = X' + T_v$$

In order to calculate T_v* we used Synge's equation for the pressure at any point in the membrane and from it found the average total pressure acting on a cross-section of the membrane taken at right angles to the root surface. As this force acts tangentally along the root and the bone it acts across the fibers and puts them under tension.

Thinking of the membrane as a suspension cable of a thickness equal to the slant length of the root, a width equal to the average circumference of the root and a length equal to the thickness of the membrane we calculated the average tension in the fibers from formulae by Dr. D. B. Steinman[8]. It was then necessary only to take the vertical component of this tension to obtain T_v.

As the tension in a cable varies quite considerably with variations in the amount of sag, tensions in the fibers for different amounts of sag or slack in the fibers were calculated.

Table II gives f and ΔL in mms and T_v and X in kgs. ΔL is the increase of L, the length of the stressed fiber, over l_1 (making no allowance, however, for elastic elongation since the modulus of elasticity and the cross-section area of the fibers are unknown). $\%\Delta L$ is the percent ΔL is of l_1. %X is the percent T_v is of X and %X′ is the percent T_v is of X′ (35.5 kg).

*Because of the slope of the fibers and the variation in pressure along the root they are not uniformly loaded. But because of their shortness this fact is considered of minor importance.

*See appendix.

TABLE II

f	T_v	X	%X	%X′	ΔL	%ΔL
.025	6.6	42.1	15.6	18.5	.0240	4.9
.010	15.6	51.1	30.5	44.0	.0212	4.3
.005	29.2	64.7	45.0	82.5	.0208	4.2

It will be noted that as the sag f decreases due to decreasing waviness of the fibers, their tension increases rapidly and with it the part of the load they support. Thus the load required to depress the tooth a given amount is greater than that based on the incompressible membrane theory alone. Or, stated another way, for a given load the depression of the tooth and the pressure in the membrane are less than for the incompressible membrane theory alone. They are less by the percent shown in the fourth column of Table II.

DISCUSSION

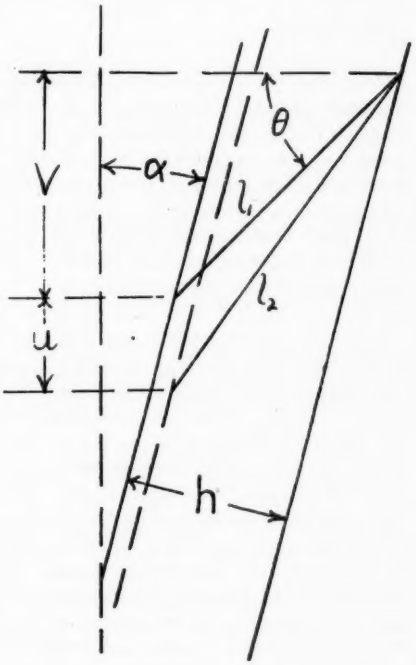
It seems probable that the fibers alone could support the tooth except for two conditions, 1) their waviness and 2) the fact that the membrane consists of other tissues than just the fibers.

The waviness of the fibers would increase the stress in them due to the effects of dynamic loading unless the pressure required to express the vascular blood were sufficient to cause a slow rate of loading. This does not seem to be the case except for a very small load.

There is, therefore, no apparent reason under the generally accepted theory of tooth support, for the fibers to be wavy but an obvious reason why they should not be.

While the load was limited to an axial force the same principles apply to the transverse load. Synge showed that for such a load the pressure is maximum at a point between the center of the tooth and the marginal ridge of the socket on the side toward which the tooth moves and on the opposite side near the apex. Opposite these points of high pressure are points of negative pressure (tension). At the apex, the middle of the tooth and the gingival margin the pressure is atmospheric under a purely transverse load.

In the incompressible membrane-fiber support theory the fibers have more slack as the tooth approaches the socket than their waviness alone would allow. These fibers are therefore not put under tension. On the opposite side of the tooth the fibers tend to become straight as the tooth moves away from



the socket. At the same time, due to the negative pressure the curves of the catenaries are convex toward the point of highest negative pressure so that the fibers exert a pull assisting the positive pressure on the opposite side of the tooth.

A theory is strengthened or weakened by the results of controlled experiment. In this respect the experiments of Mühlman and Mühlman and Zander are of special interest. The results, unfortunately for numerous reasons, cannot be analyzed mathematically but can be compared in a general way with what could be expected theoretically.

The larger displacement which Mühlman[2] calls "initial tooth mobility" corresponds very well with what could be expected during the forcing out of the blood. The "secondary tooth mobility" corresponds to compression of the periodontal membrane. The interesting point here is that multiplying the force by 5 gives approximately ⅓ the movement of the initial stage. Erupting teeth showed a greater initial mobility than functioning teeth due to greater vascularity.

Mühlman[3] finds that after multiple continuously repeated measurements on tooth mobility the mobility increased to a certain point and then remained constant within a quite definite range. This is to be expected in compression of the membrane because interstitial fluid would slowly be forced out and osmotic pressure exceeded. It is difficult to explain on the fiber support basis.

Mühlman and Zander[4] found that when teeth were moved labially the labial bone was elastically distorted which indicates pressure, while the lingual width of the membrane is not increased beyond a certain point. They ascribe the latter to the fact that the extensibility of the collagenous fibers is practically negligible. Schmitt[6] states that the extension of tendons may be as much as 10–15% before rupture. Mühlman and Zander found that in well functioning teeth the increase in thickness of the membrane on the tension side was "between 3.3 and 4.5 percent only." Synge's theory and the incompressible membrane-fiber support theory would explain this as a result of the negative pressure and negative pressure plus fiber tension, respectively, in the membrane which would be as effective in pulling the lingual bone with the tooth as the positive pressure on the other side would be in distorting the labial bone.

Mühlman's[5] experiments with artificially altered periodontal membranes have little of interest. The results are not unexpected. His postmortem findings are what would be expected with continued expression of fluid from the tissue and no physiological function to restore it.

SUMMARY

The theory that the tooth is fully supported by the collagenous fibers of the periodontal membrane falls short, with mathematical analysis, on numerous points and does not satisfactorily explain the results of controlled experiments.

The incompressible membrane theory of Synge is satisfactory in all respects except that it does not take into consideration the fact that the distribution and arrangement of the collagenous fibers indicate a function other than to render the membrane anisotropic. It also requires rather high pressures for biting forces within the normal range.

The combined fiber and incompressible membrane theory attributes to the fibers the function of partial support by tension in them caused by the pressure within the membrane forcing the tissue tangentially. This has the net effect of increasing the area at the root. Thus a lower pressure is required to support a given load.

The waviness of the fibers permits some sag in the stressed fibers and thus reduces their tension which might otherwise be excessive. Because tangental displacements within parts of the membrane are much greater than normal displacements the fibers are put under tension with the first minute movement of the tooth.

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APPENDIX

If X' is the axial load or resistance due to pressure of the membrane and $T_v = T \sin \theta'$ (where θ' is the angle the fibers make after the depression of the tooth) the resistance due to tension of the fibers, the total load or resistance X is given by

$$X = X' + T_v$$

Synge[11] gives the resistance X' of the axially loaded tooth in terms of the displacement u as

$$X' = 3/2\ \pi\mu u_2 a^4 b_1^{-3}, \sec \alpha \tan^3 \alpha$$

where μ is the shear modulus, a the length of root, b_1 the thickness of the membrane after displacement of the capillary blood and α is the half angle of the root. u_2 is the displacement of the root after expression of the capillary blood and accounts for the pressure in the membrane. $b_1 = b - u_1 \sin \alpha$ where b is the original thickness of the membrane and u_1 is the displacement necessary to express the capillary blood.

For the excess p' of the pressure p in the membrane above atmospheric pressure P Synge[12] gives

$$p' = p - P = -3\mu u_2 b_1^{-3} \sec \alpha \tan \alpha (a^2 - x^2)$$

in terms of u_2. (The minus sign denotes pressure as against tension and is ignored).

If A is the area of a section of the membrane perpendicular to the surface of the root at x distance from the apex we may write

$$A = \pi \cos \alpha\ [(x \tan \alpha + b')^2 - x^2 \tan^2 \alpha]$$

where $b' = b \sec \alpha$ whence

$$A = \pi b' \cos \alpha\ (2x \tan \alpha + b')$$

The total pressure in the membrane at any point x is given by

$$p'A = 3\mu u_2 b_1^{-3} \tan \alpha\ (a^2 - x^2) \cdot \pi b'(2x \tan \alpha + b')$$

If we denote by A_c the area under the curve between $x = o$ and $x = a$ we may write

$$A_c = 6\pi b' \mu u_2 b_1^{-3} \tan^2 \alpha \int_0^a (a^2 - x^2)\ x dx + 3\pi b' \mu u_2 b_1^{-3} \tan \alpha \int_0^a (a^2 - x^2) dx$$

Integrating between the limits $x = o$ and $x = a$

$$A_c = 3\pi b\ \mu u_2 b_1^{-3} \tan \alpha \left(\frac{a^4}{2} + \frac{2}{3} b' a^3\right)$$

Dividing by a we obtain the average value of $p'A$ and substituting the value of b' we obtain

$$(p'A)_{AV} = 3\pi\mu u_2 b_1^{-2} \sec \alpha \tan \alpha \cdot \left(\frac{a^3}{2} + \frac{2}{3} b_1 a^2 \sec \alpha\right)$$

This is the average total pressure W throughout the membrane. If we now think of the membrane as a suspension cable of a thickness equal to the slant length of the root, of a width equal to the average circumference of the membrane and of a length equal to its thickness b_2 we can write

$$w = \frac{W}{b_2}$$

where w is the pressure per unit length measured across the membrane. $b_2 = b - (u_1 + u_2) \sin \alpha$.

It is necessary to find l_2 the length of a straight line joining the points of attachment of a fiber when the tooth is completely depressed and also the new angle θ this line makes with the horizontal. Let this angle for the unloaded tooth be θ. If u is the total displacement

$$l_2 = [(V + u)^2 + (V \tan \alpha + b \sec \alpha)^2]^{1/2}$$

and $\theta' = \sin^{-1} \frac{V_4 + u}{l_2}$

where $V = \frac{b \sec \alpha \tan \theta}{1 - \tan \alpha \tan \theta}$ (See figure.)

(For the unloaded tooth l_2 becomes l_1 which is $V \csc \theta$ The value l_1 is used only to calculate δ for Table I.)

D. B. Steinman[8] gives the tension T along the cable of a suspension bridge at the extremity corresponding to the end of the fiber attached to the tooth as

$$T = H \left[1 + \left(\tan \phi + \frac{4f}{b_2^2}\right)^2\right]^{1/2}$$

where H is the horizontal component of the tension in the fiber, ϕ is the angle the chord makes with a perpendicular to the slope of the root and is equal to $\theta' + \alpha$ and f is the sag or slack. The sag is measured parallel to the root surface from the center of the closing chord of the curve to the curve.

$$H = \frac{wb_2^2}{8f}$$

The approximate length of L of the curved, stressed fiber is given by Steinman as

$$L = b_2 \left(\sec \phi + \frac{8}{3} \frac{b_2^2}{f \sec \phi}\right)$$

The results shown in Table II were obtained for the three values of f by taking $u_1 = 0.025$ mm, $u_2 = 0.005$ mm, $\theta = 45°$, $\alpha = 14.5°$, $\alpha = 12.4$ mm, $b = 0.250$ mm, $\mu = 53 g/mm^2$.

The following additional values were found:

$X' = 35.5$ kg $\theta' = 47°$

$l_1 = 0.492$ mm $b_2 = 0.243$ mm

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Necrotizing Ulcerative Gingivitis, a Reportable Disease?

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As early as by the end of the last century the question of the communicability of acute necrotizing ulcerative gingivitis was hotly debated.[1] During and after World War I this disease was widely accepted to be communicable.[2] In recent years scientific opinion seems to have moved away from this assumption so that today a majority of investigators and writers believe that necrotizing ulcerative gingivitis is not communicable.[3-11] Many writers[2, 5, 8, 10, 11] and Committees[6, 7] have based their conclusions as to the non-communicability of necrotizing ulcerative gingivitis in part or largely on one study.[3] It is, however, important to note that Schwartzman and Grossman,*[3] the authors of this study, like other medical workers of that period,[12, 13] while speaking of Vincent's stomatitis, apparently dealt with herpetic gingivostomatitis.[5] At no time do Schwartzman and Grossman[3] give an exact clinical description of the condition they discuss. Of 9,706 pediatric patients examined they found 33 cases of so-called "Vincent's ulcero-membranous gingivostomatitis." Ten of these cases were observed in infants during the second year of their lives, the other 23 reported by them in children up to 7 years of age. No cases of what these workers called "Vincent's" were found by them in their entire series of examinations in any of their patients of

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the ages 7-13. They reiterate that this condition is much more frequent in children than in adults and state that no local therapy of any sort tested appreciably changes the time for a cure from between 13 to 15 days. These findings and statements indicate that what these workers called "Vincent's" were in fact cases of herpetic gingivostomatitis. The observation of smears positive for the Fusobacterium plauti-vincenti and for the Borrelia vincenti as reported by those authors cannot, in the opinion of this writer, be taken to give even suggestive validity[5] to their finding of non-communicability, as far as necrotizing ulcerative gingivitis is concerned, because such organisms may be present[2] or act as secondary invaders in a wide variety of oral lesions including acute herpetic gingivostomatitis.[13,14] Schwartzman and Grossman's conclusions[3] should, therefore, be considered not pertinent and entirely non-conclusive for the question of communicability of acute necrotizing ulcerative gingivitis.

There are some authors that feel the disease is communicable,[15,16] or that the question of communicability is, at least, not yet clearly resolved.[17,18]

The question of communicability is naturally of greatest importance for the decision whether a disease should be reportable to the health authorities of the State.

In 1944 Fowler compiled data on "Diseases and Conditions Required To Be Reported In The Several States."[19] In this he mentioned 22 states in which "Vincent's infections (Vincent's angina; acute Vincent's infection)" was reportable. In 1945 Sebelius[20] elaborated on Fowler's work as it pertained to Vincent's infection.

The writer thought that after a time lapse of 10 years it might be interesting to note how the State Health Departments now felt as to the reportability of necrotizing ulcerative gingivitis and the associated stomatitis and pharyngitis. For this reason the following inquiry was sent to the Health Officers of the 48 states and of the District of Columbia: "Will you kindly inform me whether any of the following are reportable in your State by physicians or dentists: Vincent's infections (necrotizing, ulcerative gingivitis), Vincent's stomatitis, or Vincent's angina."

RESULTS OF INQUIRY

1. Two States, Illinois and Washington, replied that the mentioned conditions were reportable within their jurisdiction.

2. One State, Tennessee, replied, that while these conditions were reportable at this time, they were to be *definitely* dropped from the list of reportable diseases at the forthcoming revision of their regulations.

Two States, Indiana and Kansas, replied that while these conditions were reportable at this time, they would *probably* be dropped at the forthcoming revision of their regulations.

One State, Colorado, reported that while these conditions were reportable at this time, they were *probably* to be dropped at the forthcoming revision of their regulations except for reporting of cases in an "explosive outbreak."

3. Thirty-seven States and the District of Columbia replied without qualifications that none of the diseases mentioned were reportable within their jurisdiction.

4. Two States, Rhode Island and Texas, require reporting for Vincent's angina only, not for necrotizing ulcerative gingivitis.

5. Three States, Missouri, Oregon and Utah, do not require reporting of any of these conditions; however, in case of an epidemic they require their reporting as in case of any other disease.

DISCUSSION AND CONCLUSIONS

It is clear that the question of the communicability of necrotizing ulcerative gingivitis cannot be settled by means of questionnaires. It should be mentioned, too, that reportability of disease depends not

only upon communicability, but also upon other factors, as, for instance, seriousness of the disease and whether enforcement of reportability is feasible. Yet the results of such an inquiry are significant because they permit the diagnosis of a trend within both the dental and the medical professions as to their opinion concerning this important phase.

From the answers received it may be seen that out of 49 Health Departments of all States and the District of Columbia *only six States consider isolated routine cases of necrotizing ulcerative gingivitis as reportable; four of these six have signified their intent to drop such cases from the list of reportable diseases in the near future.* Compared with Fowler's[19] and Sebelius[20] study of 10 years ago the long range trend as of today is clearly toward the dropping of necrotizing ulcerative gingivitis from the list of diseases that must be reported to the State-, and District of Columbia-, Health authorities.

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An Evaluation of Topical Terramycin in Postgingivectomy Pack

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THIS study was made to determine the effects of terramycin on the periodontal tissues following gingivectomy. The study was instituted as an attempt to evaluate the rational of incorporating terramycin in the surgical pack following gingivectomy. It is desirable for the wound to heal with a minimum of discomfort. Terramycin, placed in the postgingivectomy pack, may have certain potentialities to aid healing. Whether or not there is a sound basis for using it in this phase of periodontal therapy must be studied and analyzed.

REVIEW OF LITERATURE

In the literature there are many reports on the systemic use of terramycin in oral infections and in oral surgical procedures.[1-6] These reports state that excellent results were obtained in the use of systemic terramycin and very few side reactions were manifested by the patients. The authors placed very little importance on the allergic reactions that were observed in the cases treated with terramycin.

Krause, Johnson and Casey[7] reported a controlled study in which they found an apparent synergistic effect of saliva with terramycin in vitro. This study used serial dilutions of terramycin in Penassay broth and serial dilutions of the antibiotic in saliva-broth. Bacillus cereus, Bacillus mycoides and Betahemolytic streptococci were the organisms used in the experiment and control. Bacillus mycoides was used to determine a median bactericidal end point. The results showed that, in the presence of saliva, less than two-thirds the amount of terramycin produced the same effect as the full amount of terramycin in the broth alone. The authors demonstrated that the important variable was the saliva-to-terramycin ratio rather than the absolute concentrations of either. It was clearly shown that the addition of 0.63 volume percent of saliva increases the median bactericidal effect of the antibiotic by 100 per cent.

Shpuntoff and Shpuntoff[8] reported on a study of the prolonged activity of topical terramycin in vitro. The antibiotic for the experimental work was incorporated in a surgical pack. The object of the experiment was to determine the amount of terramycin liberated by a standard pellet immersed in the tubes of broth for specified periods of time. The broth was Difco dextrose broth. Three tubes, with 10 milliliters of broth in each, were used for each determination. Two loopsful of stock cultures of Staphylococcus aureus (Columbia stock culture) were used. These cultures were incubated for eight hours at 37° C. after which they were placed in a refrigerator at 5° C. until used to inoculate the tubes. The authors concluded that terramycin mixed with zinc oxide-eugenol was stable and active. The antibiotic was slowly released into the broth and saliva-broth. It was found to be bactericidal at the end of five days.

Many questions regarding terramycin and its use in dentistry are unanswered. Some of them are as follows:

1. Does terramycin mixed in the surgical pack aid in healing following gingivectomy?

2. What is the degree or rapidity of epithelization of the area of gingivectomy when terramycin is mixed in the pack?

3. What are the subjective symptoms of the patient in regards to discomfort, pain, and odor?

4. Will the terramycin reduce or prevent the odor after the pack has been in the mouth for several days?

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The opinions or assertions contained in this article are the private ones of the writer and not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

5. Are there any local allergic manifestations from topical terramycin?

6. Will patients who manifest allergic reactions to the topical terramycin have allergic reactions to systemic terramycin, at a later date?

7. Will patients who manifest allergic reactions to topical terramycin have allergic reactions if the topical terramycin is used again, at a later date?

8. Will those patients who did not have any reaction to topical terramycin manifest an allergic reaction the next time topical terramycin is used?

The following study was undertaken to answer some of the above questions and to conduct a controlled evaluation. Patients were selected for this study from those which had been scheduled for routine gingivectomy.

EXPERIMENTAL PROCEDURE

Fifty patients were treated in the following manner in this study. The postgingivectomy surgical pack utilized as the experimental included 125 milligrams of powdered terramycin in 6 drops of liquid (eugenol 98% and thymol 2%) to which sufficient powder (Box's formula) was added to produce a very stiff mix. The postgingivectomy pack used as the control was identical in composition and consistency except terramycin was not included. On each patient a gingivectomy was done on the maxilla or mandible. A surgical pack was placed immediately after gingivectomy. The cases used in this study had nearly the same periodontal involvement on both the right and left sides of the maxillary or mandibular arches. One quadrant of either the maxillary or the mandibular arch was the control and the other quadrant, of the same arch, was the experimental. Comparisons were made only between quadrants of the same arch. The quadrants were either full or partial and the same number of teeth or nearly the same number were included in the gingivectomy of both the experimental and the control sides. The packs for both the control side and the experimental side were kept in place approximately the same length of time in the individual patient. This variable was reduced as much as practical in keeping with good therapy. The time the packs remained in place varied in different patients. The patients did not know that there was any difference in the packs used.

Careful observations and comparisons were made as to the degree and rapidity of epithelization of the surgical wound on the experimental side and the control side in each individual case. An objective check of the packs was made for odor, before and after removal. The subjective symptoms of the patients that were recorded included discomfort, pain, and odor. Any abnormal reactions from either the control pack or the experimental pack were recorded.

Histological sections made from the biopsy material obtained from one patient were studied microscopically to determine whether or not the epithelization was more rapid under the experimental pack or under the control pack. A gingivectomy was done on the labial and buccal surfaces of six teeth on both the experimental and control sides, but the tissue was not bevelled. The packs were placed immediately after gingivectomy. They were removed in sections as the biopsies were taken. Biopsies were taken on both the experimental and the control quadrants of the patient. A pair of biopsies (1 control and 1 experimental) were taken on the seventh, tenth, fourteenth and eighteenth day after gingivectomy. The biopsy excisions were made in such a manner as to result in a bevelling of the tissues. The tissues were fixed in formalin, sectioned, and stained with hematoxylin and eosin.

A small segment was removed from the postgingivectomy surgical pack and was placed on a sterile blood agar plate with approximately 2 c.c. of the patients saliva. This plate was then incubated at 37° C. The plate was read after 48 hours of incu-

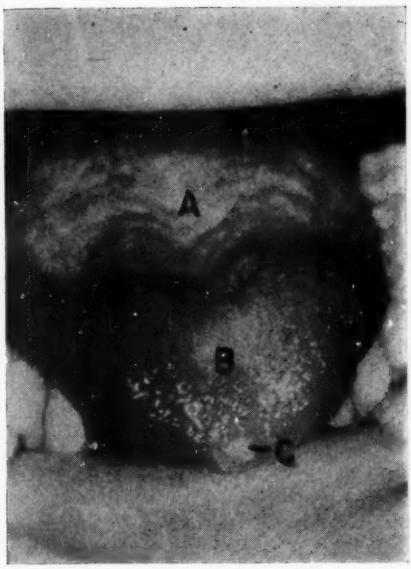

Fig. 1. An oral manifestation of allergic reaction. A—Black hairy surface of tongue. B—Erythematous tip of tongue. C—Vesicle on tip of tongue.

bation to determine the relative zone of inhibition around the segment of the pack. This procedure was repeated on both the experimental and the control sides on the seventh, tenth, fourteenth and eighteenth day after gingivectomy. There was a ten day interval between the use of the control pack and the experimental pack. This was intended to give some indication as to the effectiveness of the packs in preventing bacterial growth at the time intervals described. The original packs were used throughout the procedure.

Oral terramycin was administered to eleven of the patients, who had allergic manifestations, after the idiosyncrasies had completely disappeared. They were given 500 milligrams every four hours for three days, a total of 9000 milligrams. This was to determine whether or not allergic manifestations to topical terramycin go hand in hand with allergic reactions to systemic terramycin.

On four of the patients who had the most pronounced allergic reactions to topical terramycin, it was again used when gingivectomy was done on the other arch. This was to determine whether or not they would again manifest reactions to topical terramycin.

In ten cases that did not exhibit allergic reactions, topical terramycin was used in both of the remaining quadrants of the opposite arch when the gingivectomies were done. This would determine whether or not there would be reactions to topical therapy upon repeated use. The gingivectomy and packs with terramycin were repeated at approximately two week intervals.

RESULTS

In forty-eight of the fifty patients, clinical observations revealed that the healing was more rapid on the side where terramycin had been incorporated in the pack. The epithelium covered the healing wound at a more rapid rate on the side in which terramycin had been used.

Of the fifty patients, forty-nine indicated that the side with the terramycin was more comfortable. The other one did not have an opinion and did not feel pain with either pack.

All fifty patients were positive that one pack had a bad taste and definitely more odor than the other pack. In each instance this pack was the one which did not have terramycin.

There were no allergic reactions to the control pack, but twelve of the fifty patients manifested an allergic reaction to topical terramycin in varying degrees. Four of the twelve patients had a marked reaction while the remaining eight ranged from moderate reactions to only an objective symptom of a black hairy tongue. Although the terramycin pack was placed on only one quadrant at a time the allergic manifestations were always bilateral. In these cases with allergic reactions the tissue under and immediately adjacent to the terramycin pack did not exhibit any of the allergic manifestations. The

buccal mucosa, tongue and soft palate were the sites in which allergic manifestations were observed (Fig. 1 and 2).

Eleven of the patients, who had allergic reactions to topical terramycin were given oral terramycin (500mg. q4h for 3 days). These eleven patients did not manifest any allergic reactions to parenteral terramycin therapy.

The four patients, having the most pronounced reactions, were given topical terramycin therapy again after the original idiosyncrasies had completely disappeared. Three did not have any abnormal reaction to the antibiotic. One manifested the same type of reaction as was initially observed.

In ten of the thirty-eight patients which

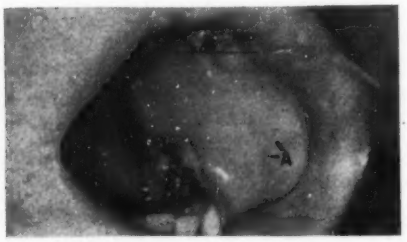

Fig. 2. An oral manifestation of allergic reaction. A—Erythematous patches in buccal mucosa.

did not have any reactions to topical terramycin, it was used again, at approximately two week intervals in both the remaining quadrants. In these ten cases there were no allergic reactions to the repeated antibiotic therapy.

The tissue removed by the initial gingivectomy revealed an epithelial layer with marked acanthosis, normal keratinization and a well defined basement membrane. There was a moderate round cell infiltration of the connective tissue.

In the sections taken periodically after the initial gingivectomy the epithelium, which was proliferating to cover the wound and the epithelium adjacent to the wound, revealed a marked vacuolization of the

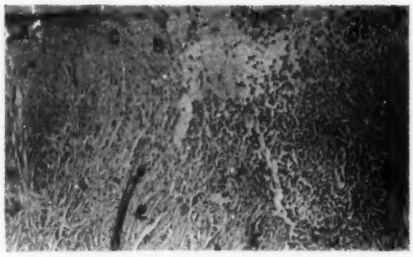

Fig. 3. Section from experimental side on seventh day. A—Old epithelial layer. B—New epithelial layer. C—Vacuolization of epithelial cells and intercellular edema. D—Granulation tissue. E—Artifact.

epithelial cells and intercellular edema (Fig. 3 and 4). This condition was found in the control for a longer period of time than in the experimental (Fig. 5 and 6). Vacuolization of the epithelial cells and intercellular edema was not found in the experimental side on the fourteenth day, but was still discernible on the control side on the eighteenth day (Fig. 6).

A beginning of rete peg formation in the new epithelium was observed on the experimental side on the fourteenth day. Definite rete peg formation was evidenced on this side on the eighteenth day (Fig. 5). It was not until the eighteenth day that the beginning of rete peg formation was observed in the new epithelium on the control side (Fig. 6).

On the tenth day the beginning of a differentiation between the basal layer and the prickle cell layer could be found in the new epithelium on the experimental side. This differentiation was very slight on the

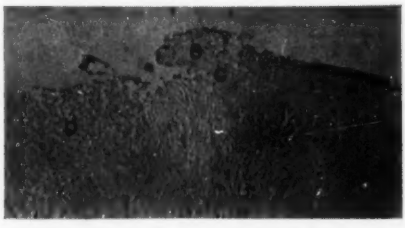

Fig. 4. Section from control side on seventh day. A—Old epithelial layer. B—New epithelial layer. C—Vacuolization of epithelial cells and intercellular edema. D—Granulation tissue.

Fig. 5. Section from experimental side on eighteenth day. A—New mature epithelial layer. B—Well defined prickle cell layer. C—Well defined basement membrane and well defined rete peg formation. D—Very little round cell infiltration.

control side on the eighteenth day. (Fig. 6).

The new epithelial layer on the eighteenth day appeared to be somewhat thicker and more mature on the experimental side than on the control side (Fig. 5 and 6).

There was marked round cell infiltration of the underlying connective tissue on both sides on the seventh day (Fig. 3 and 4). This round cell infiltration gradually decreased on the control and the experimental sides at about the same rate as healing progressed. However, on the eighteenth day there was less round cell infiltration in the connective tissue on the experimental side than on the control side (Fig. 5 and 6).

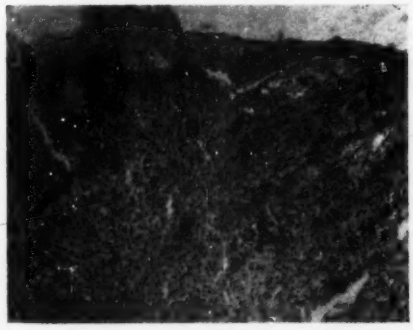

Fig. 6. Section from control side on eighteenth day. A—Thin immature new epithelial layer, with slight differentiation of prickle cell layer. There is no definite basement membrane on this section. Very slight tendency toward rete peg formation. B—Marked round cell infiltration.

There was some endothelial proliferation, but this finding seemed to be more prominent in the experimental sections (Fig. 3 and 5).

The blood agar plates showed a definite zone of inhibition around the experimental pack as compared to a very narrow indefinite zone of inhibition around the control pack which were removed from the mouth at the end of seven days (Fig. 7 and 8). At the end of ten days the zone of inhibition exhibited around the control pack was much less than that around the experimental pack (Fig. 9 and 10). There was

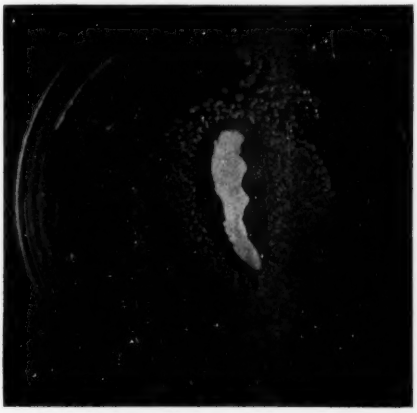

Fig. 7. Sample of experimental pack removed at the end of seven days.

no zone of inhibition around the control pack removed at the end of fourteen days (Fig. 12). The zone of inhibition evidenced around the experimental pack removed at the end of ten days is wider than that exhibited by the experimental pack on the seventh or the fourteenth day (Fig. 9, 10, 11 and 12).

DISCUSSION

The findings were definitely confirmed that the patients were more comfortable, noticed less odor and experienced less unpleasant taste with the packs containing terramycin than those without terramycin. Results indicate the packs containing

terramycin were a superior therapeutic procedure to those not containing the antibiotic as evidenced by the rapidity of epithelization.

It is interesting to note that all manifestations of allergic reactions were found bilaterally in the oral cavity. The tissues manifesting the allergic reactions were not in contact with the pack containing the antibiotic. The tissues in contact with the antibiotic pack exhibited no reaction. In every instance it was observed that the allergic reactions did not retard healing, but conversely, these cases healed rapidly. Although the reactions were undesirable, it appeared the cases with the more pronounced allergic reactions healed more rapidly than those with less pronounced reactions or with no reaction at all.

In my opinion the cases which manifested a reaction to topical therapy, can be rationalized on the following basis. In the topical application they were receiving a very small dosage. A small dose may produce an irritating effect, whereas, the systemic dosage maintained a high blood level which did not create an irritating effect. Hence no allergic reactions were manifested in the systemic therapy. The lack of any reaction to oral administration by these patients would suggest that the topical use of terramycin does not necessarily sensitize the patient to the systemic use of this antibiotic. It is further suggested that although a patient has a reaction to topical terramycin at one time, a subsequent reaction to topical therapy may not always occur. It is possible there is some factor other than the mere application of the topical terramycin that enters into the etiology of the allergic reaction.

A study is indicated to determine the influence of the organisms remaining after some of the strains have been destroyed by the terramycin. The altered oral flora and the imbalance enforced by the terramycin may predispose to allergic reactions. It is entirely possible the filamentous organisms, that may be present after other forms have been destroyed by the antibiotic, have a very different effect upon the oral tissues when they are no longer in the presence of the usual oral flora.

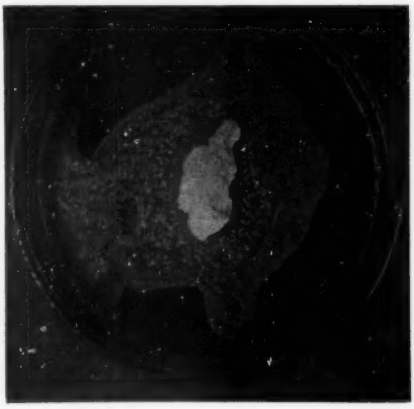

Fig. 8. Sample of control pack removed at the end of seven days.

The average time for the clinical manifestations of the allergic reaction to appear was approximately four and one half days. This time lag before the onset of the manifestation of the allergic reaction is much longer than the usual time necessary to determine allergic reactions to specific materials in routine allergy tests. This time lapse

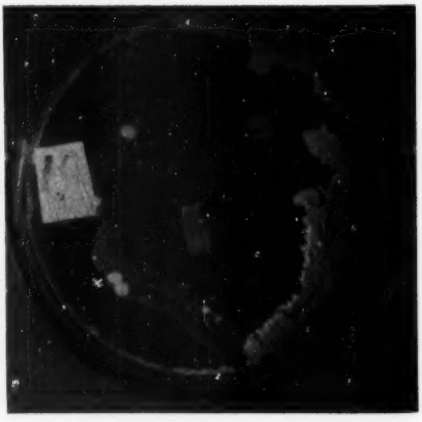

Fig. 9. Sample of experimental pack removed at the end of ten days.

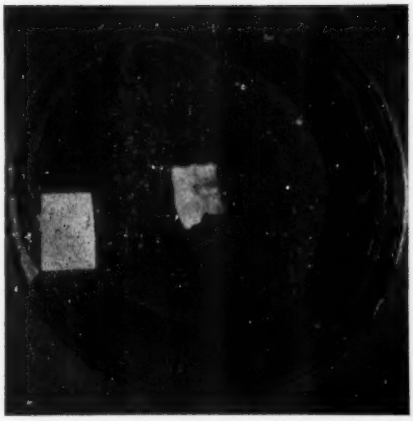

Fig. 10. Sample of control pack removed at the end of ten days. Numerous areas appear as artifacts due to bubbles in saliva.

would allow for the growth of the remaining cultures of organisms to such an extent that they or their products could be the cause of the reactions.

The histologic study demonstrated that the epithelization of the wound under the experimental pack was more rapid than under the control pack. This histological finding was borne out in clinical observations when comparing the rapidity of healing of the experimental side with the control side.

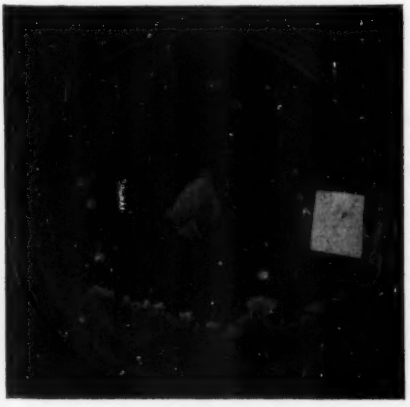

Fig. 11. Sample of experimental pack removed at the end of fourteen days.

The zones of inhibition found on the blood agar plates indicated that the effectiveness of the antibiotic was greatly reduced by the fourteenth day. Therefore, if surgical packs are to remain longer than this, fresh packs should be placed. Ideally the terramycin packs should be replaced on the tenth day to insure the maximum benefit of the antibiotic. It is also my opinion that if terramycin is not used in the packs they should be replaced on or before the seventh day to prevent a foul odor, bad taste, and discomfort.

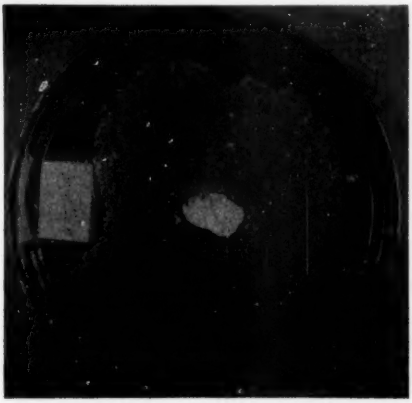

Fig. 12. Sample of control pack removed at the end of fourteen days.

SUMMARY AND CONCLUSIONS

Of the fifty patients treated with postgingivectomy surgical packs containing terramycin, twelve manifested allergic reactions during the period the antibiotic packs were in place. Four of these twelve patients manifested a pronounced reaction while these packs were in place. The remaining eight patients experienced reactions that ranged from moderate to only the symptom of a black hairy tongue. The onset of these allergic manifestations averaged four and one half days after the beginning of the topical therapy. Reactions to repeated use of the antibiotic topically, whether the patient had a previous reaction

or not, were found in only one patient. There were no allergic reactions to the control packs in the fifty patients treated.

The bilateral manifestations of the reactions and the lack of any manifestations of a reaction in the tissues under or adjacent to the pack containing terramycin indicates these reactions were not from direct contact with the antibiotic.

Systemic terramycin does not necessarily produce allergic reactions in patients that have previously exhibited allergic reactions to topical use of terramycin.

The zones of inhibition found on the blood agar plates were most pronounced around the terramycin pack on the tenth day. These zones were diminished on the fourteenth day and markedly reduced on the eighteenth day. The zones of inhibition were very narrow around the control pack on the seventh day. The zones of inhibition indicate that the packs containing terramycin should be changed on or before the tenth day to insure the best inhibitory action of the bacteria by the antibiotic in the pack.

This study demonstrated that the use of terramycin in postgingivectomy surgical packs makes the patient more comfortable, eliminates nearly all of the odor and unpleasant taste of the pack, and shortens the healing time. The epithelization of the wound area appeared to be more rapid under the terramycin packs during the eighteen day period microscopically and macroscopically.

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POETRY (?) CORNER

Georgie Porgie, pudding and pie,

Kissed the girls and made them cry.

The reason of course is now quite clear,

His breath smelled of garlic, tobacco and beer!

Psychosomatics in Relation to Periodontics*

by JOSEPH S. LANDA, D.D.S.,† *New York, N. Y.*

PSYCHOSOMATICS is now one of the most timely topics of discussion at medical and dental conferences. Both professions are devoting a significant portion of their interest to the thorough revival and implementation in daily practice of the health principle long ago formulated by the ancient Romans in their celebrated dictum: "Mens sana in corpore sano." A sound mind in a sound body. That the psychosomatic concept of medicine was also known to the ancient Greek physicians is clear from Plato's comment on the error his contemporaries were making in trying to divorce the mind from the body. Neither was Hippocrates unmindful of the interrelationship between the psyche and the soma. He emphasized the importance of *temperament* and prescribed physical and mental rest in the treatment of disease.

The tendency to see only organic disease and to view the soma completely dissociated from the psyche dates back about two centuries. It was about that time that physicians began to manifest great interest in dissections in order to better observe the ravages of many diseases throughout the body. Later on histology came to the fore. The microscope brought into view astounding microscopic tissue changes. This was followed by tremendous progress in the field of bacteriology with discoveries about bacteria, viruses, fungi, etc. which contributed considerably to more effective treatment and prevention of organic disease. Last but not least "came the marvels of surgery and what Osler called the discovery of the pathology of the living: that is the discovery of the frequency with which abdominal pain is due to organic disease such as acute appendicitis, cholecystitis or peptic ulcer" (Alvarez).

So great and widespread was the enthusiasm over the discoveries made in the realms of organic and physical disease that physicians completely lost sight of the psyche. It did not occur to them for a long time that the very peptic ulcer so spectacularly excised by surgery, might itself have been caused by emotional disturbances. Nor did it occur to them that surgical wounds heal better and sooner in a happy patient than in one who is mentally depressed. We now know that the psychic effect of a physical disease may be worse than the physical ailment itself. A man who has suffered a slight attack of coronary thrombosis with correspondingly very slight physical damage to his heart may become an invalid through fear and anxiety.

We know of no disease of the body that will not adversely affect the mind; conversely, any disease of the mind will affect the body in some way or other. Therefore, all diseases may be classified as psychosomatic or somatopsychic, depending upon the predominance of either the psychic or somatic component. It is true that it is frequently extremely difficult to determine with certainty which is predominant or to establish definitively which acts as the etiological factor for the other.

Such difficulty is encountered especially in procrastinated cases of undiagnosed chronic organic diseases that have become encrusted through the years with emotional disorders of various types. In the past, because of their diagnostic difficulties, such ailments were lumped together as "functional" and were either treated palliatively or not at all, particularly when the patient was suspected of being a "crank."

It is accepted today that long-continued mental strain and nervous tension may be

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the etiological factor in peptic ulcer. In the same way, a mental disorder may be caused by a long continued physical torment or by an organic disease. There is no exclusively physical or emotional disease. Both always coexist, serve each other as a background and are reciprocal in their effects.

This pertains to diseases in any part of the human body as well as to diseases in the oral cavity. No disease or lesion in this latter zone, as far as we know today, can be ascribed exclusively to psychologic etiological factors. The latter, however, are potent contributors to the precipitation and aggravation of circulatory, neurologic, infectious, nutritional, metabolic and allergic manifestations of disease in the oral cavity. This is our concept of the approach to the study of the interrelationship between psychosomatics and periodontics.

Psychogenic disturbances may lead to periodontal disease or aggravate a preexisting periodontal disturbance. Exacerbation of periodontal disease by emotional stress and strain has been observed clinically so frequently and so clearly that the relationship can no longer be doubted. Vincent's infection (acute necrotizing ulcerative gingivitis) is observed most often in adolescence, a period of life probably more plagued with psychologic problems and emotional unrest than any other. It is especially pronounced in our own time of hot and cold wars, which seem to be working havoc with family and home life. Severe emotional tension is also associated with various infatuations so characteristic of adolescence. Disappointment in love affairs frequently engenders conflicts and moral humiliation. Dissension between parents and adolescent children are so common they cannot be regarded as isolated cases. The prevalence of Vincent's infection in adolescents should, therefore, not be looked upon as a mere coincidence, but rather as a contributary cause-effect relationship. Mindful of the important role the psychologic factors play in the etiology of Vincent's infection during the period of adolescence, we nevertheless should be wary of regarding the psychological factor as the only and single cause of Vincent's gingivitis.

In the multifaceted scheme of etiology of disease the psychologic factors are only one of many that participate in bringing on periodontal disease. Vincent's gingivitis in many adolescents may be the resultant of a continuation of early neglect, the gingivitis tending to manifest itself in its severest form at that time. We should also not lose sight of the possible oral effect of hormonal changes incident to maturation. There is also the effect of drinking alcoholic beverages and smoking, usually started in early adolescence and often indulged in to excess as a new means of demonstrating sophistication. The latter, however, in itself constitutes a psychologic component. This only serves to emphasize the fact that in the living human being no iron curtain can ever separate the psyche from the soma. The two are always interwoven and in a state of continuous interplay. S. C. Miller* expresses the interplay between the psyche and soma in relation to periodontal disease in the following manner:

"It can be stated with fair assurance that in periodontia the psychosomatic relations can be revealed with more clarity and in many instances almost with mechanical certainty. The three broad categories of psychosomatic relations may be listed thus:

1. Periodontal diseases causing disturbances of the psyche;
2. Psychogenic factors causing or aggravating periodontal disease;
3. Continued effect in both directions. The last is the usual finding in most cases unless seen at a very early stage."

Let us now analyze the situation with a view to finding out to what extent the psychosomatic and somatopsychic phe-

*The Dynamics of Psychosomatic Dentistry—Joseph S. Landa. Dental Items of Interest Publishing Co. Brooklyn, N. Y., 1953.

nomena may influence the periodontal structures in the direction of health or disease. To achieve this, we must examine the cultural environmental conditions under which we live and develop. Under a given culture the evolution of the individual is channelized along three main pathways.

1. Biological
2. Psychological
3. Technological

There is very little an individual can do to influence the progress of his own biological evolution, since this is mainly under the control of a certian set of natural laws. The other two—namely, psychological and technological can be vastly influenced by the individual. The sky rocketing advances in technology in our time are frequently working against man's own interests. Thus the advances in the technological treatment and preparation of our foods have so far exceeded the biological evolution of the masticatory mechanism that a serious gap is here manifest between the capacity of the structures and the functional demand upon them. The foods in our culture are so prepared as to afford contemporary man limited opportunity for vigorous use of his masticatory apparatus. The inadequate use of this natural function combined with emotional factors which are always present in some form or another have an adverse effect upon the dental structures in several ways:

1. Rampant caries;
2. Alveolaclasia with rapid bone resorption;
3. Or a combination of both, which is frequently the case, with each serving, secondarily as a background for the other.

The good diagnostician is interested in knowing which of the structures is primarily and predominantly involved. However, it is extremely difficult to answer this question with certainty and it is just as difficult to establish the exact mechanism by which emotional disturbance and inadequate use of the masticatory apparatus cause periodontal disease. The reason for this difficulty is twofold:

1. No disease, trivial in nature as it may be, is ever caused by one factor—there is always a multiplicity of factors participating together in the etiology of any disease.

2. These factors usually exert their influence in the direction of disease in an indirect way. Thus the causative medium might be an alteration of the salivary constitution with possibly some alteration of secretion from glands of the mucous membrane. An alteration of the quality, quantity and consistency of the oral secretions could, in turn, adversely affect the oral flora. It is logical to assume that these changes might permit proliferation of bacteria on the gingiva and in the gingival crevice, this leading directly to a gingivitis. We can assume with a great degree of probability that the same etiological factors are at work in preparing a fertile soil for rampant caries. However, whether their selectivity is greater for caries than for periodontal disease we do not know. The correct answer will help us not only to treat the disease more effectively but perhaps also to reduce its incidence considerably. It may also indirectly shed light upon the etiological factors of excessive alveolar bone resorption under artificial dentures.

We know, on the other hand, that the health of the periodontal structures and the firmness of the teeth in their sockets depend upon the following conditions:

1. The proximity of the lamina dura to the root of the tooth.
2. The height of the alveolar bone;
3. The status quo of the bone factor.

All these structural features depend upon hereditary factors, proper function of the masticatory apparatus and systemic influences of which we know very little.

The periodontal structures are highly sensitive to *inadequate stimulation and overstimulation.* We find that the present cultural environmental conditions exert an adverse effect upon the masticatory mechanism in both directions:

1. Understimulation—because our current soft foods require little or no mastication.

2. Overstimulation — because of the prevalence of neurosis leading to bruxism, nail-biting, pencil-biting, tooth-pick-biting, etc.

The damaging effect of neurosis on the periodontal structures is not limited to these forms of mechanical overstimulation alone. Neurosis also has far-reaching adverse effects upon the general nutrition and indirectly lowers the resistance of the periodontal structures. McCall has seen many severe cases of Vincent's gingivitis in children in the 5 to 10 year age range with indications that malnutrition was the principal causative factor. I have had the experience of treating many victims of severe Vincent's gingivitis in lands that were ravaged by famine. We witnessed most spectacular cures in 3-4 days by supplying the victims with adequate amounts and proper quality of food. The importance of nutritional factors in the maintenance of good health is too well known to require here more elaboration. However, psychologic factors exert considerable influence on nutrition indirectly and in doing so adversely affect the health of the periodontal structures. All the psychodynamic avenues of approach indicate that periodontology surely should include study of the psyche as well as the soma.

Patients suffering from severe and long-continued emotional strain soon become victims of indigestion followed by disturbed metabolism. This, in turn, may be and usually is reflected in the oral cavity by gingivitis, an inflamed mucosa, alveolar resorption and other forms of periodontal disease. Digestion does not depend only upon what one eats; the state of mind while eating is also a factor. Fear anxiety and mental depression may lead to a reduction of glandular secretions, including the glands of the digestive system, and thus considerably diminish the potency of the agents of digestion and assimilation. The circulatory system is also highly sensitive to and adversely affected by emotional disturbances. A breakdown in any part of this transportation system must be followed by impairment of function and disease in various parts of the body. To this effect, the periodontal structures are no exception. In other words, proper nutrition and assimilation are impossible unless the circulatory system and the systems of secretion and excretion (urine, sweat, etc.) are functioning properly. Their unimpaired function is a pre-requisite for a healthful body chemistry. But the proper function of the body chemistry is no less dependent upon stable emotions than it is upon proper blood circulation.

The effects of the psyche upon the periodontal structures may thus be regarded as the consequence of

1. circulatory changes
2. structural changes

The effector agents which induce these changes from health to disease are

1. Internal agents
   a. hormonal agents
   b. neurological stimulation
2. External agents
   a. direct trauma
   b. inadequate function
   c. excessive function

In both instances, the internal and the external agents, it is the psyche which participates in inducing the disease. Thus:

1. The classic involuntary vagal response to stress overtly indicates the relationship of the psyche and the soma.

2. The functional habits of the patient are not accidental but rather psyche motivated and directed and the periodontal dis-

ease is precipitated as the consequence thereof.

For this reason the psychosomatically-minded dentist, unlike his mechanically-minded predecessor, gives much more thought to the interrelationship between the general health of an individual and the health of his periodontal structures. Similarly, he ties up emotional with physical health, by giving careful consideration to the emotional components of the patient's personality. The dentist should not only bend every effort to comprehend the patient's needs, drives and anxieties but should go a step further in an endeavor to learn about the psychological content of the patient's emotional status. The oral cavity, which is the beginning of the alimentary canal, is controlled, like the rest of it, by the sympathetic and parasympathetic systems. Stimulation of the sympathetic system brings about hyposecretion, whereas stimulation of the parasympathetic system produces hypersecretion. In health, the sympathetic and parasympathetic systems are in perfect balance. Emotional unrest and strain disorganize the autonomic nervous system and frequently manifest themselves in the oral cavity by xerostomia, hyper-salivation, disturbed circulation and a metallic, bitter taste. All of these symptoms may be and frequently are associated with gastro-intestinal diseases of various kinds, and represent the end result of emotional strain. The good diagnostician cannot fail to observe clinically the adverse effect these syndromes manifest in the periodontal structures.

Disease, as we know, is usually due to multiple factors, though of varying potency. It would, therefore, be a grave mistake to attribute periodontal disease to a single local or systemic cause. Many systemic and many local factors are at work simultaneously, and, superimposed upon one another they may lead to an end-result called periodontal disease. Since this disease is frequently fatal to the teeth, its *prevention* is the best cure. The earliest signs of aberrations in the periodontal structures should be diagnosed and treatment directed toward elimination of all possible etiological factors. The health service in this branch of dentistry is not limited to saving teeth or to preventing focal infection which often resides in periodontal pockets of even moderate depth. *Preventive periodontics* may, in addition, have a most salutary effect upon our emotional and psychological health.

It is a well known fact that periodontal disease (pyorrhea alveolaris) is a most dreaded affliction. It holds more terror for the patient than any other dental disease. First because "pyorrhea" is always associated in the patient's mind with the inevitable loss of all the teeth. Secondly, the dread of focal infection from the "circulating pus" symbolizes to them the infirmity of every imaginable systemic disease. Some patients are so sure of their own diagnosis that they demonstrate with their finger to the dentist the course the pus pursues from the dental alveoli to the shoulder or hip joint—wherever they may happen to have the pain at the time. We cannot and should not brand them as psychotics or even as "cranks." In most instances these are people like the rest of us but they happen to live in environmental conditions highly charged with emotional situations. Its victims usually make the rounds from dentist to dentist in quest of a promise of a complete cure. If we take into careful consideration such patient's concern and mental distress, we readily understand the vicious circle in which he is trapped.

We have observed clinically many cases in which deteriorating gingival conditions accompany the patients' severe emotional disturbances which involve his or her family life. Improvement in the latter situation frequently reflects correspondingly in improved gingival condition. It follows that the periodontist and the general practitioner must at all times be aware of the psychosomatic implications in this specialty of dentistry. A blunt announcement to the patient that he has pyorrhea may very well plant a seed of torment in his mind, thus

considerably aggravating the very illness for which he seeks treatment. The thoughtful dentist will make the patient aware of his condition in a careful and tactful manner so as not to cause emotional trauma.

But here we come vis-a-vis the problem of doctor-patient relationship, one of cardinal importance in any and all therapeutic procedures. We, as dentists, are all fully aware of the dangers facing us when using sharp separating disks in the posterior region of the mouth. We are also fully conscious of the danger of opening into the maxillary sinus when endeavoring to remove root apices in the region of the upper first molar. Few of us, however, realize that our general behavior and our way of speaking to the patient may be fraught with more harm to him than accidental opening into the Antrum of Highmore. Improper rapport and lack of understanding between doctor and patient will not only render the treatment less effective but may wreck the patient emotionally by causing irreparable psychic trauma. To calm a panicky patient, to put him at ease and to win his complete confidence is both an art and a science. In addition the dentist must be highly skilled and competent in his work. He must be highly ethical in his practice and possess a high degree of sensibility to the suffering of others.

It is these qualifications of the dentist that infuse implicit faith of the patient in the doctor. Out of this faith emerges a doctor-patient rapport which, like a perfectly woven fabric, is integrated in perfect balance and mutual trust and understanding. By careful and tactful history taking we first get acquainted with the patient's emotional background, his health problems and cultural milieu. It is then and then only that we can speak to the patient on his own level and hope to effect a meeting of our two minds. Failing to understand the patient's emotional and cultural background we would run the risk of projecting our own concepts and values on a patient who might not in the least be receptive to them. This would result in lack of cooperation and continued misunderstandings between doctor and patient. When doctors prescribe diets, regulation of exercises or hobbies for patients, they take too much for granted when they believe that the patients interpret them or attach to them the same meaning and significance as the doctor does. Such assumption on the part of doctors renders their therapy ineffective in many instances, and in others may lead to distress because of misinterpretation by the patient.

Patients cannot all be treated in the same way even for the same condition, because each one represents a distinct personality pattern. Each one's personal capacity for appreciation and interpretation of various phenomena in life depends upon his upbringing, his state of physical health, his ambitions, his likes, his dislikes and his way of evaluating values in life. The doctor cannot afford to *depersonalize* the patient's personality by treating him just like a chart number. In undertaking the treatment of a patient for periodontal disease, the dentist must not only be thoroughly versed in diagnosis of the mechanical and biological phases involved but, in addition, must have a fair familiarity with the psychological background of his patient. Prior to the appearance of the patient in his office, the patient may have visited other dentists or may have been *adversely conditioned* to dental treatment by friends and relatives who have shared their experiences with him. In order for the treatment to be successful, such a patient must first be *deconditioned*.

This does not mean that we should *at any* time undertake to play the psychiatrist's role. This would, indeed, be very hazardous for the patient and dentist alike. It does, however, mean that we should strive to acquire an elementary knowledge of psychology and steadily exert every effort to *cultivate* a feeling of sympathy and compassion for all our patients. They trust us with the most precious thing in life—their health. In return we are to give them not only the highest type of dental

health service from a mechanical and biological standpoint (which is really our primary calling) but also the emotional support they so badly need.

A basic knowledge of human psychology and the application of good common sense (not psychiatry) will help us in understanding the nature of the patient's emotional disturbance. This, in turn, will enable us to calm down a panicky and, at times, even a despondent patient by permitting him to lean on us with his full weight. Such psychotherapeutic procedures may restore the acutely disturbed emotional equilibrium of the patient by encouraging the action of his reasoning faculties, so essential for warding off the momentary threatening situation. For the treatment to be effective, absolute confidence of the patient in the doctor as well as mutual sincere respect between doctor and patient are imperative. Confidence in the doctor bolsters the patient's hope, reinforces his confidence in ultimate recovery and thus contributes to the patient's peace of mind and happiness. *Happiness and general physical health are reciprocal and stimulate each other.* To maintain the periodontal structures in good health a person must be physically and mentally sound. Herein lies the relation between psychosomatics and periodontics.

SUMMARY AND CONCLUSIONS

For a long time dentistry was considered a mechanical specialty. In recent decades the profession became partly conscious of the importance of biologic principles in the successful management of dental operations. At present the specialty of dentistry is becoming three-dimensional in status—psycho-bio-mechanical.

Each one of us should endeavor, whenever possible, to learn the basic principles of psychology related to medicine and dentistry. Conferences, post-graduate courses and the current medical and dental literature on the subject of psychology can enrich our knowledge of this new and most fascinating approach. It is extremely important for the dentist of today to be able to recognize the mildly neurotic or borderline psychotic patient so that he may use good judgment in deciding when to direct a patient to a psychiatrist. The story is told of a surgeon who was operating surrounded by a number of assistants. At one point in the operation he suddenly and abruptly withdrew the scalpel. When asked by one of the assistants to identify by name the anatomic structure that was endangered in that particular area, the surgeon uttered into the surgical mask: "I do not know its name but I know darn well to keep away from it." This should be very instructive to us. By learning how to recognize the emotionally involved patient and even the normal patient who happens to be temporarily in a state of severe mental unrest it may prove of tremendous help to us in diagnosis as well as in treatment planning. In a normal patient, who happens to be in a state of severe mental unrest, no complicated dental procedures such as complete mouth rehabilitation should be undertaken. The impact upon the patient of the stress and strain thus experienced while subjected to the dental drill and other unpleasant episodes may, at times, serve as a precipitating cause for a serious mental disturbance.

Surgical dental operations should be avoided in patients who may happen to be near a neurotic break. The extraction of a tooth may be "the straw that breaks the camel's back." Under these and similar conditions it may also even be desirable to postpone radical periodontal treatment until the patient's emotional state has improved to a satisfactory point. When a practitioner becomes psychosomatically minded it does not mean that he pays less attention to precision technological procedures in dentistry. The more aware we become of the potential psychic trauma from improper operative procedures, the greater the drive to practice dentistry of the highest quality from a mechanical, biological as well as psychological standpoints.

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Herpetic Stomatitis

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PROBABLY the most common infections of the oral-systemic complex with manifest signs in the mouth are those of herpetic origin. Every doctor, in the course of his practice, will see herpes lesions more frequently than any other. The early diagnosis and treatment of these "canker sores," as they are sometimes called, will enable the practitioner to serve his community better by alleviating the painful symptoms invariably associated with this infection. In this paper, therefore, I propose to develop a concise picture of the etiology, epidemiology, pathogenesis, diagnosis and recent advances in the treatment of herpes simplex infections as they pertain to the oral and peri-oral tissues.

Epidemiology. We can divide herpetic stomatitis into two forms as seen clinically: primary or acute herpetic gingivostomatitis and secondary or chronic recurrent herpetic gingivostomatitis. The acute form is a generalized systemic infection; the chonic form is not. These shall be discussed separately.

Herpetic gingivostomatitis is an infectious disease caused by the herpes simplex virus, and is so widespread that it is said to occur in three-fourths of the adult population.[1] Primarily, the most hard-hit victims of the acute form are young people. Manifestations of the primary form of herpetic disease occur in children whose ages range from a few weeks to fourteen years, although by far the greatest number of cases occurs in the one to three year old age group.[2] In a study of cases at Children's Hospital and the University of Pennsylvania Hospital, Scott et al. states that though most attacks are seen in children of the ages twelve months to twelve years, forty per cent of the cases occur in children under three years of age and seventy per cent in youngsters under six years.[3] Burnet's study indicates that the greatest incidence occurs in the six months to twenty-four months age group.[4] As far as sex and racial incidence is concerned, there seems to be no significant correlation, and the conclusion reached by most authors is that there is no apparent difference in the proportion of negroes or whites affected and as many males as females are stricken.[5]

The disease is spread by direct exposure (at least in forty per cent of the cases)[6] or contact. The literature contains a description of a one year old child who was innoculated with fluid from the vesicle of a herpes simplex lesion of an adult.[7] This produced a typical attack of acute herpes simplex gingivostomatitis in the child. There are also a number of healthy carriers of the disease amongst our population who harbor the virus in their saliva, since the saliva contains the herpes virus during and between attacks. If a susceptible person comes into contact with a convalescent recovering from some form of herpes simplex infection other than the infectious stomatitis, (that is, herpes genitalis or herpes labialis) it it still possible for him to contract the stomatitis from that carrier.[8] The most frequent source of infection for children is adults—parents who carry herpes-neutralizing antibodies in their blood. Mothers are one of the endemic foci of infection for children and may infect their offspring, for, although they are not active carriers, herpes simplex virus has been isolated from the saliva of many during the menstrual cycle. However, this may work both ways. Scott et al. reports a case in which a mother, who was herpes-free as a child, contracted it from one of her children.[9]

The Virus. The etiological agent of the disease (both forms) is the herpes simplex virus. It is 0.15 microns in diameter and can be grown in the embryos of hens' eggs and in suitable laboratory animals (that is, mice, rodents, etc.). In the early 1930's,

some researchers believed that such virus infections were the result of some organism generated within the human body. However, since Dodd, Johnson and Buddeigh succeeded in isolating the virus from the saliva of infected children, the viral etiology has been pretty well established.[10]

The virus can be transmitted in saliva and other secretions of the body during the clinical course of the disease. This is especially true in the one to three year old age group.[11] In most cases, the virus enters the body through an infection of one sort or another, and it is usually not in itself apparent at the time of this infection.[12] For example, in a common cold, the symptoms of the cold mask the presence of the herpes virus upon its *initial* entrance into the body of a susceptible individual. When the cold or other infection clears up, the herpes virus remains with the patient all through life. When subsequent colds appear, the virus manifests itself usually in the form of "cold sores" or "fever blisters."

Pathogenesis. The pathogenesis of the virus has been very adequately described by Anderson.[13] In a study carried out in 1940, she used chick embryos to study the passage of the virus through the host. She traced the virus through the embryo chicks by a series of subcutaneous, subamniotic, membranal and scarification methods of innoculation. The findings that follow are chiefly the results of these tests.

The virus invades the epithelium upon contact, multiplies in the cells and produces a cellular hyperplasia. It spreads also to contiguous tissues but the means of spread is not known. The virus invades the bloodstream, and disseminated throughout the body, causes metastatic lesions. In this way, an infection from a local site may result in a generalized herpetic infection. Many of these lesions brought about by metastatic extension via the bloodstream can be prevented, at least in part, by intravenous injections of herpetic antiserum into the host. Another interesting fact is that the virulence of the organism seems to increase through successive passages through the chick embryo hosts. This virulence is especially notable for endothelium and other "mesodermal elements," as well as for liver and cardiac muscle cells. However, since the rate of infection is to a great degree dependent upon the susceptibility of the tissues involved, it is safe to assume that the liver, endothelial cells and heart cells are particularly susceptible to herpes simplex infection.

There is a third method of spread; this is neural. The virus seems to have an affinity for nervous tissue, and since the neurons and perineural cells are derived from the same germinal layer, the same tissue affinities hold here as in other methods of spread. In the central nervous system (through intercerebral and cord innoculation) the spread is from axon to axon along the peripheral nerves resulting in an ascending encephalomyelitis and a descending myelitis.[14] To summarize, then, we have three methods of pathogenesis: (1) invasion of the epithelium by direct contact, (2) hematogenetic spread which brings on metastasis and a generalized herpetic infection, and (3) ascending or descending neural infection. Metastasis and localization are proved by the fact that inclusions are found in the capillaries and endothelial cells of the large blood vessels. The most susceptible tissues to herpes attack are embryonic cells of the pharynx, peritonium, skin, amnion, liver, heart, smooth and striated muscle, lung, spleen, kidney, adrenal glands, submucosa of the gastrointestinal tract, central nervous system and peripheral neuron system, osteoblasts and chondroblasts.

Antigen-Antibody Reactions. After infection by the herpes simplex virus, type specific neutralizing antibodies are found in the bloodstream. This antibody titer increases during convalescence.[15] It has been postulated that from sixty-five to ninety per cent of all adults have herpes simplex neutralizing antibodies in their blood.[16] Those people not subject to recurrences are those without serum antibodies against herpes. Recurrences appear in people with

high antibody titers.[17] Although the body builds antibodies against the virus, it remains throughout life, since it grows *within* the cells and, therefore, it cannot be destroyed by the antibodies.[18] However, in vitro, herpes simplex virus is quickly neutralized by these type specific antibodies.[19] A question which still remains unanswered today is, why there is so great a quantity of the virus in the vesicle of recurrent herpetic infection when the antibody titer is so high.

The antigen-antibody reactions are the basis for a test to determine if patients have been infected with the herpes simplex virus. The reliability of the test or the extent of its use is unknown to the writer at the present time. The test material is obtained by innoculating the yolk sac of a chick embryo with the virus of herpes simplex. Within seventy-two hours after the innoculation, a high titer of virus appears in the amniotic fluid. This is relatively protein-free and can, therefore, be used for the actual test on human beings. The test consists of injecting the inactivated amniotic fluid antigen intradermally. After forty-eight hours a tuberculin-like reaction (positive test) is seen on the skin site of the injection in patients who have been infected with the virus. A control is also run. This consists of injecting only normal amniotic fluid.[20]

Character of Herpetic Lesions. The lesions of all herpetic infections are similar. It is very difficult to distinguish between the lesions of the acute, primary and chronic, recurrent forms, if it is possible at all. However, we may say that, as a general rule, in the recurrent form the lesions are larger, and deeper ulcers are formed.

Usually the early lesions are described as "elevated areas of tissue composed of a fibrinous exudate within the epithelium . . . . (there is a) modified round cell infiltration in the subjacent connective tissues."[21] There are marked changes in all the layers of the epithelium and a secondary infiltration of inflammatory cells. The epithelial cells are swollen and exhibit a "ballooning" and reticular type of degeneration. Intranuclear inclusion bodies, characteristic of many of the virus diseases, are found within the cells of the primary lesion.[22] These inclusion bodies were first reported by Lipschutz in 1932, and therefore bear his name.[23] Within the nucleus, these inclusion bodies stain eosinophilic; a ring of chromatin collects at the margins of the nucleus and nucleolus. The nucleus also has a homogeneous ground glass appearance.

As seen clinically, the lesions are round or oval vesicles which vary in size from a pinhead to several millimeters in diameter. The vesicles are filled with a clear, yellowish, watery fluid containing the herpes simplex virus and degenerating cells. The vesicle is formed by intracellular edema and hydropic degeneration of the epithelial cells; later, some PMN's appear in the fluid and corium as well. The stage of the vesicle formation is relatively short, especially if the lesion is in the mouth; the trauma during eating and the maceration in the saliva cause it to rupture forming shallow ulcers that are typical. These ulcers immediately become secondarily infected and an exudate is produced which contains much fibrin, PMN's, and cellular debris. The submucosa undergoes simple necrosis and becomes infiltrated with the leukocytes. Around these eroded areas very prominently seen are bright red areas of inflammation sharply demarcated and giving the appearance of a crimson halo. The lesions are very painful, and difficulty in eating is frequently experienced combined with an increased flow of saliva. Many times the ulcers will coalesce to form shallow, large, irregularly shaped ulcers, and around the periphery of these ruptured vesicles are found small pieces of epithelium which are the remnants of the ruptured wall. Within a few days, healing takes place with a yellow, thick cheesy material forming over the surface of the large, ulcerated areas. After ten to fourteen days the lesions disappear. Scarring may occur, but only in cases where there

are repeated secondary infections at the site of original lesions.

Sometimes Vincents fusospirochets are found; however the herpes simplex virus is always found both in the smears and vesicular fluid. It has been definitely shown that the Vincent's organisms are not the cause of the disease although frequently seen.[24] No immune antibodies are found in the early stages, but occur in varying numbers in convalescing patients.

Herpangina. Some authors have reported a clinical entity called herpangina with symptoms similar to that of herpetic gingivostomatitis.[25,26] They have postulated that the etiological agent is the coxsackie virus. However, there must be more work done on this subject and more proof presented on whether this is a definite and separate disease, or merely a form of herpetic manifestation. The lesions are vesicular in nature and the disease is a fibrile one. Age, occurrence, frequency and site of the lesions (mainly on the fauces) are so similar as to be not merely coincidence. A definite statement, however, cannot be made at this time.

Clinical Signs and Symptoms of Primary Herpetic Stomatitis. Primary or acute herpetic gingivostomatitis has been confused with many other diseases. It has been called aphthous stomatitis, acute ulcerative stomatitis,[27] catarrhal and membranous stomatitis,[28] periadenitis mucosa, necrotica recurrens, chronic intermittent recurrent aphthae (Mickulicz) or solitary and recurrent Mickulicz type,[29] fusospirochetal stomatitis, trench mouth, and it has been confused with Vincents' angina or Vincents' stomatitis.[30]

The acute form of herpetic gingivostomatitis manifests itself as an acute systemic infection. It is generalized and often severe due to the fact that the victim has no protecting antibodies. At times it is preceded by another condition or disease process which lowers the hosts' resistance to the virus. The most frequent of these is an upper respiratory infection; but often implicated as predisposing factors are infectious mononucleosis, gastro-intestinal disturbances, a common cold, meningitis, pneumococcal pneumonia or severe trauma. Frequently the onset is insidious and presents non-specific symptoms which may be overlooked. This is especially true in infants who may drool excessively, refuse to eat or drink and cry without apparent reason. In a series of cases studied by Scott et al., the trouble was not located by the mother for from two to four days.[31] Older children present symptoms such as lethargy, irritability, refusal to play with their friends, fretfulness, quarrelsomeness, headache, pain on swallowing and cervical adenitis. Diarrhea is sometimes a symptom; in such cases the patient is acutely ill.

In articulate patients, a sore mouth is the most frequent complaint for twenty-four to forty-eight hours preceding the appearance of the vesicles. The prodromal symptoms are a very high rectal temperature, ranging from ninety-nine to one hundred six degrees Fahrenheit, accompanied by submental, submaxillary and cervical lymphadenopathy.[32] The fever subsides usually upon the appearance of the ulcers or within seventy-two hours thereafter. Sometimes the clinical picture is complicated by dehydration and acidosis.

In the clinical stage, the gingiva is tender and swollen (sometimes almost covering the teeth) with a tendency to bleed easily.[33] The severity of the disease determines the number and size of the vesicles. The vesicle, which immediately upon rupture is clean, is covered by a greyish pseudo-membrane and becomes extremely painful. Fetor oris is always present in cases of primary herpes simplex stomatitis. The most common areas involved are the buccal mucosa, tongue, gingival and inner surface of the lips; however, the floor of the mouth, trachea, tonsils, esophagus, larynx, soft palate and even nasal mucous membranes may be involved. The prodromal stage lasts from one to two days and ends with the eruptions of the vesicles. The lesions frequently appear in bunches or crops every twelve to four-

teen hours. The vesicles rupture in two to twelve hours; the ulcers form and last five to ten days after which spontaneous healing takes place usually without scarring.[34] Lymphadenopathy persists, however, for as long as two to three weeks after symptoms have disappeared. The disease produces a moderate degree of immunity—herpes neutralizing antibodies are noticed in the blood stream and a severe recurrent crisis rarely occurs. However, the disease in its generalized or acute form is quite serious and has been known to cause death, especially in the very young.[35] Paronychia, impetigo and meningitis are a few of the complications which may result.

Clinical Signs and Symptoms of Secondary Herpetic Stomatitis. Recurrent herpetic gingivostomatitis is the most common form of the disease. It occurs usually in older people who have some humoral resistance to the infection—those who have been infected previously, although many cases do not give a history of primary infection. Appearance of the lesions and etiology are the same for the recurrent as for the acute form. The trigger mechanism is unknown; however it is thought to be any one of a number of factors which tend to lower tissue resistance and consequently, allow the intracellular virus to produce the disease.

In most cases, a specific flaring factor is present. This may be an allergy, gastrointestinal disturbance, trauma, menstruation, exposure to bright sunlight, fever, upper respiratory infection, hypnotic suggestion, common cold, influenza, seasonal variations, thyroid disease, pregnancy, emotional crises or psychosomatic disturbances. In short, any condition which, by any method, reduces local tissue resistance and/or increases the metabolism of the body cells may cause a recurrence of the herpetic infection.

In this disease, since it is not as severe, one does not get the generalized symptoms of the primary form. Instead, a feeling of "fullness," or an itching, burning sensation is noticed at the site of the future lesion —these are prodromal symptoms. One or two days later the vesicles appear, followed by rupture, ulcer formation, healing and disappearance without scarring in from five to ten days after the vesicles first appear. Clinical symptoms include sialorrhea and lymphadenopathy. The lesions occur repeatedly in the same locations owing to the intracellular character of the herpes simplex virus when it first enters the body. The disease is self-limiting and runs its course. Although the lesions are painful, this form of herpes infection is not serious.

It is interesting to note in detail what specific factors can precipitate an acute exacerbation of the symptoms in this form of the infection—those principles spoken of as the flaring factors above.

Allergy. Hypersensitive reactions to food are known to produce recurrent aphthae. Walnuts are especially incriminated, although it has been found that other foodstuffs may also be associated with this trigger mechanism. Oatmeal and chocolate are frequent producers of these outbreaks.[36] It has never been definitely proved, however, that those so affected were not allergic to the foods previously or that they were not hypersensitive to other things which might have brought on symptoms. Another interesting case is that of the pregnant woman in whom ulcers re-occur continuously during the period of gestation. At parturition the attacks cease. To my knowledge, no comparative studies have been made on women who exhibit pregnancy tumors of the gingiva and those who suffer from recurrent herpes simplex infections during the term of pregnancy.

Of interest to the dentist are those who are victims of herpes attacks after dental operations. It has been known for some time that patients on whom a rubber dam is placed, or who have undergone oral surgery, sometimes return to the dentist with a score of painful aphthae, seeking his help. Trauma in operative and crown and bridge work frequently is the trigger

mechanism producing local, usually single, lesions on lips and at the reflection of the labial or buccal mucosa.

Recently, the psychosomatic aspect has received a great deal of attention. Bland and Brady, in a study of some ten patients with recurrent oral aphthae, attempted to treat them by suggestive therapy.[37] The patients had had extensive medical care and treatment for recurrent herpes simplex infections although no obvious cause for the occurrences were determined. They presented the following evidence:

1. There seems to be a correlation between a feeling of guilt or shame and an outward visible manifestation in the form of repeated attacks of herpes simplex.

2. A specific trigger or flaring factor which brings on these attacks was demonstrated in the subjects tested.

3. The patients were immature, rather hysterical, "highly suggestable and overreactive to small stimuli." They were given psychiatric treatment which they readily accepted, and a "rapid transference" developed between the psychiatrist and patient. This was due they reported, to a high degree of "infantile sexuality."[38]

4. "Psychoanalytically they (the patients) would be classified as oral receptive people in whom the attacks of herpes appeared as a negative response."[39]

5. In all cases except one, an immediate relief of symptoms was found. Follow-up studies revealed that this was not temporary, but lasted for the entire length of the study (months).

6. In two patients, a reappearance of symptoms came on within twenty-four hours. This was precipitated by a break in the "interpersonal relationship" between patient and psychiatrist through an abrutly cancelled appointment.[40]

7. In these people, erotic desire with frustration seemed to bring on the attacks. "Psychologic conflict was expressed predominantly in oral terms."[41]

8. One case presented in detail was that of a twenty-seven year old white woman who was sexually promiscuous, and, although no conscious feeling of shame was evident, she did react afterwards in each instance with recurrent attacks of herpes.

9. "The specific mechanism for herpetic eruptions seems to be a return of repressed oral impulses, in orally receptive people, a projection of oral sadistic tendencies with guilt and shame for erotic feelings. The physiology of the mechanism whereby the skin reacts to the feeling of guilt and shame is unknown."[42]

A change in the weather is said to be a possible flaring factor responsible for recurrent herpes simplex aphthae about the oral cavity; some authors have stated that the greatest number of recurrences appear in the winter.[43] However, since the number of colds and upper respiratory infections occur in the cold months, and since winter weather taxes normal individual resistance to the utmost, it is probable that this is tied up with systemic disease as a possible trigger.

Therapy. Although there is no known specific treatment, many have been proposed for both the primary and secondary forms of this disease. I shall discuss these, and leave the practicability of their use to individual clinical judgment.

The disease is unalterable in its three stages—prodromal, active and convalescent—and does not respond to curative measures. The treatment, therefore, in primary herpetic infection is supportive and symptomatic. Steigman et al. used one per cent gentian violet applied locally or one to two doses of chromium tri-oxide.[44] In this report, the lesions and visible mouth signs disappeared in from six to sixteen days (or an average of eleven days.) Alkaline mouthwashes for the relief of acute symptoms and removal of debris are generally agreed to be helpful. Kerr proposes dissolving one-half teaspoonful of salt and one teaspoonful of baking powder in one quart of warm water.[45] The patient is then

given directions to irrigate the mouth with one quart of this solution each hour for several hours until relieved. Chance secondary infection is reduced by swabbing the mucosa with two per cent acriflavine, 1:500 nitromersol or mercocresol. Antibiotic troches may be used to help reduce bacteria in the mouth and therefore cut down the chances for secondary infection. Aspirin can be used as an antipyretic to reduce the fever, forced fluids to prevent dehydration and acidosis. Parenteral fluids may be given subsequently if necessary in cases of extreme dehydration. Aureomycin has been tried to reduce duration of the disease without much success.

In treating the chronic recurrent form, local caustics have been used (such as trichloroacetic acid, silver nitrate and phenol) to reduce pain. Penicillin and aureomycin troches are used by some clinicians to reduce secondary infection. Oral lavage with warm alkaline mouthwashes (as above) have also been proposed.[46] Braitman has tried aureomycin capsules (eight) of 250 mgms. each.[47] Patient was given the directions to take one in the morning and one in the evening. He reported seemingly excellent results. Within two days, pain disappeared, and within the next two days (four days after the onset) the lesions were gone. This patient was free of lesions for the first time in seven years. The lesions did not return until six weeks later. She was given the same treatment, continued only for sixteen days, and to the date of writing of the article (five months later) the patient has had no recurrence.

Woodburns suggests prophylaxis in the form of smallpox vaccination.[48] This form of treatment has been advocated by many other authors and has been fairly successful. Six repeated injections of smallpox vaccine in increasing doses has been tried by the University of Michigan with very excellent results.[49] Investigators have also advised the use of snake venom to prevent recurrence.[50]

Those symptoms on an allergic basis are treated with antihistaminic drugs. Those on a psychosomatic basis can be treated with excellent results by suggestion therapy by a competent psychiatrist. For lowered tissue resistance, Burket proposes vitamin B complex therapy.[51] He obtained amazing results with this form of treatment. He prescribed one to five milligrams per day of thiamine hydrochloride, and the lesions all but disappeared within ten to twenty-four hours after treatment. Supplemental diet regulation was used concurrently. In a few cases, five milligrams of thiamine hydrochloride were given about the time the vesicle was about to rupture. The medication was taken at bedtime, and by morning the ulcers were gone and no pain existed. Subsequent administration of five milligrams of thiamine hydrochloride for several days eliminated all fibrous areas at the vesicle site.

Some researchers have tried such things as yeast tablets (patients improve within twenty-four hours, it is maintained),[52] nicotinic acid,[53] riboflavin,[54] silver nitrate and zinc chloride,[55] alum and camphor ice,[56] camphorated phenol,[57] neoarsphenamine,[58] sulfonamides,[59] Roentgen rays,[60] formolized herpes virus injections of autogenous serum,[61] intradermal snake venom,[62] and smallpox vaccination.[63]

As you can see from the above, the present status of treatment for both these conditions is in a state of flux. Palliative and supportive therapy are the wisest plans of treatment, with possible thiamine hydrochloride therapy as an adjunct.

Herpetic stomatitis has been confused and misunderstood for many years. In putting together the pieces, I hope that I have helped to clear up some of the confusion that has existed heretofore.

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Let's Follow the Trend*

by EDWARD L. BALL, D.D.S., *Cincinnati, Ohio*

TODAY, thanks to science, we have a far better chance for a long, healthy life than people had who lived in the past century. By immunizing the well and quarantining the sick, physicians guard us against communicable diseases. The purity of food we eat and the milk and the fluoridated water we drink is carefully protected. To encourage healthful measures all those interested in physical welfare provide every possible practice to prolong life.

Medicine today is concerning itself with a program of Geriatrics. Why? The reason is obvious. With a prolonged life span certain ideas become mandatory in treating individuals. It follows that with longer living there must be new methods, new approaches, new procedures to cope with the new situation. With these needs presenting themselves Medicine is broadening its knowledge by research.

Recently, while attending an all day meeting of Geriatrics it occurred to me that if medicine is interested in the treatment of the aged so must dentistry—an integral part of medicine—be alerted to the treatment of our "New Citizens of Longevity."

One formerly hesitated to refer to others as old or aged because somehow it suggested "the end." This condition no longer exists. Everywhere about us we hear of those who reach the 90, 95, 100, and even past the century mark. Today there are more than 13,000,000 people at the age of 65 than there were in 1946.

Isn't it wonderful news to know that we are going to live longer, to enjoy more years of youth. Then let us glorify the word "old" with new hope and new vigor. Let us follow the trend. Let us help make "our old people" our respected "Senior Citizens," or our "Grandis Population."

What part is dentistry to play in this new program? It follows that we, the dentists, must further our work by unearthing some new facts that may aid us to retain longer our natural teeth. If then by chance there are those among this "Grandis Group" who are unfortunate in losing their teeth then let us be ready with newer, better and more thorough procedures for replacing them.

Dentistry must keep abreast of these developments. It must come to the front with better and newer methods of treatment. Our laboratories and our departments of research must forge forward finding new methods, developing new techniques and above all testing new scientific truths.

To be able to grow old with one's natural teeth should be sheer satisfaction not mentioning the sheer delight and superb accomplishment. According to the Bible in Psalms, 90th Chapter, Verse 10:

"The days of our years are three score years and ten."

May we hope to say by reason of developed methods of dentistry that the days of our years are four score years and ten, and perhaps by reason of more and greater strength they may become five score.

3268 Jefferson Ave.

*Read before the Blue Grass Dental Society, Feb. 25, 1956, Lexington, Ky.

HAROLD KEITH BOX

May 24, 1956

Dr. Harold Keith Box, age 66, died very suddenly at his home in Toronto on May 24th, 1956. Just a few days before his death he outlined to a group of his associates, with his usual eloquence and enthusiasm, new studies that he was anxious to develop. He had spent all of the day immediately preceding his death in the service of his patients.

Since Dr. Box's childhood in Carleton Place, Ontario, his career has been characterized by academic brilliance, insatiable inquisitiveness and the courage to be progressive. These qualities were supported by a strong integrity of purpose and true devotion to his family, his friends and his profession.

In the same year (1914), that he graduated from the Royal College of Dental Surgeons of Ontario, he joined the staff as a demonstrator in bacteriology. He had a continuous association with the staff of the Royal College of Dental Surgeons and later with the Faculty of Dentistry of the University of Toronto until his retirement in May, 1956. From 1920 to 1927, he served as Professor of Dental Pathology and Periodontology. From 1927 until his death he was Research Professor in Periodontology.

He received a Ph.D. degree in 1920. In 1921 he was elected the First Fellow of the American Academy of Periodontology. Dr. Box was a fellow in Dental Surgery of the Royal College of Dental Surgeons, a fellow of the Royal Society of Medicine and of the Royal Microscopical Society of England, an honorary member of the American Academy of Dental Medicine and an honorary life member of the Ontario Dental Association and the Toronto Academy of Dentistry.

Among his many awards were numbered the Canadian Dental Research Medal, the Jarvie Fellowship Medal of the Dental Society of the State of New York and the Fones Memorial Medal of the Connecticut Dental Association. He also was honorary consultant to the Ontario Department of Health.

Of the many honours conferred upon him, he seemed to cherish particularly the occasion when The Canadian Dental Association conferred on him honorary life-membership during the May 1955 convention. A portrait of Dr. Box, presented at that time, now occupies a place of honour in the Faculty of Dentistry of the University of Toronto.

He had written a total of seventy articles, bulletins and monographs. Most of these were related to the etiology, histo-pathology and treatment of periodontal disease, but the remainder showed a wide range of interest in all matters pertaining to dental disease. His latest monograph "Oxygen Insufflation in Periodontal Disease" was published in 1955.

His imagination led him from what could have been an easy but stifling security into the realm of the strange and difficult. For over forty years he pioneered and he led us to a greater measure of perception.

C.H.M.W.

Book Notices . . . .

There have been several books published recently which deserve the careful consideration of all dentists, particularly those interested in the treatment of periodontal diseases. Of particular note are two new color atlases of oral pathology. The first is the *Atlas of Clinical Pathology of the Oral Mucous Membrane* by B. Orban and F. M. Wentz and over forty contributors. (C. V. Mosby Co., St. Louis, 1955). This book opens with an outline designed to help the student and practitioner arrive at a diagnosis in a systematic manner. It considers the various lesions of the oral mucous membranes, white lesions, ulcerative lesions, bullous lesions of the mucosa, epuli, tongue lesions and discolorations. The authors include photographs and brief discussions of the clinical features, course of the disease, differential diagnosis, etiology, histopathology and therapy or oral infectious diseases and non-infectious diseases of the oral cavity. The book includes 72 colored illustrations, 142 black and white illustrations, and 14 charts. Readers will find the book both interesting and valuable.

The second is the *Color Atlas of Oral Pathology,* prepared by the United States Naval Dental School of the National Naval Medical Center, in Bethesda, Maryland (J. B. Lippincott Co. Philadelphia 1956). This book, written largely by Captain Robert A. Colby, includes 461 pictures in color and a bibliography or 20 pages and an index. The photographs are carefully prepared and are excellent color reductions of the subject under discussion. The text describing each photograph is concise and essentially authoritative. Brief consideration is given of the histopathology of some of the conditions discussed, although most of the illustrations are of clinical photographs. The book should be an excellent supplement to a course in oral pathology.

Another book deserving study is the book entitled *Periodontal Therapy* (H. M. Goldman, S. Schluger, and L. Fox—C. V. Mosby Co. St. Louis 1956). This book, with 190 illustrations and 100 plates, is over 560 pages in length. The profuse illustrations are designed to illustrate various techniques in periodontal therapy; little space is devoted to the histopathology of periodontal disease. The authors are to be complimented on the excellence of their illustrations and the concise descriptions accompanying them.

Another book that is of interest to oral pathologists is the new edition of *Sutton's Diseases of the Skin* (C. V. Mosby Co. St. Louis, 1956). This lengthy reference book includes discussions of the anatomy, physiology and pathological changes of skin and mucous membrane. The section on the oral mucosa is short and not at all complete so the book cannot be used as a reference in oral pathology. For example, periodontitis is not mentioned in the index and pyorrhea alveolaris is discussed in 29 lines. The statement is made "Pyorrhea eventually heals itself: the gums recede, the teeth fall out, and the sockets heal with scar." It is hoped that the treatments suggested in other sections of the book are more desirable from the patient's point of view!

A second edition of *Physiological Foundation of Dental Practice* by L. L. Langley and E. Cheraskin (C. V. Mosby Co. St. Louis, 1956) has just been released. This book is designed as a textbook of physiology for dental students. The authors discuss in a lucid fashion the nervous system, circulation, respiration, excretion, aliments, and endocrinology. Students and dentists will find the book both readable and accurate. The book includes 192 illustrations, primarily drawings, and supplemental references at the end of each chapter to guide the more ambitious student in further study.

Two other reference books are worthy of mention. The new edition of W. H. McGehee, H. A. True and E. F. Inskipp's *Textbook of Operative Dentistry,* has just been released.

(Blakiston Div. of McGraw Hill Book Co. New York 1956) Periodontal problems are not considered in this text but operative dentistry is authoritatively presented, and all recognize the importance of good operative dentistry in the maintenance of normal tooth attachment apparatus.

Also recently published is a 400 page paper bound booklet entitled *Les Parodontopathies* (Tipographia Luigi Salvagno, Venezia, 1955). This book has original text and summaries in four languages—English, French, German and Italian, and includes reports and documents presented at the 14th meeting of the Association for Researches into Periodontal Diseases (ARPA Internationale) which met in Geneva in September 1955. Included are articles by C. D. Marshall-Day, L. N. Peterson and R. T. Dunkin, H. Becks, and R. Pencharz, S. C. Miller, M. N. Okum, L. J. Baume, A. W. Ward and many others. Anyone interested may order the book from the Secretary General, ARPA Internationale, 2 rue Bartholoni, Geneva, Switzerland.

Resolution adopted by the Board of Directors of the William J. Gies Foundation for the Advancement of Dentistry, Inc. on the passing of Dr. William J. Gies who died of a Cerebral Hemorrhage at his home in Lancaster, Pennsylvania on May 20, 1956

WHEREAS, The William J. Gies Foundation for the Advancement of Dentistry, Inc. has suffered an irreparable loss in the death of Dr. Gies; and

WHEREAS, for nearly half a century Dr. Gies has given of his time and his extraordinary talents to the advancement of dentistry with no thought of reward other than that of serving his fellowmen; therefore, be it

RESOLVED, that we, who have known Dr. Gies throughout his many years of unselfish service in the promotion of human welfare, join with his many friends in extending to Mrs. Gies and the members of his family, heartfelt sympathy; and, be it further

RESOLVED, that a copy of this resolution be given a permanent place in the annals of the Gies Foundation, and that copies be sent to the dental journals for publication.

The death of William J. Gies means that dentistry has lost one of its most capable, energetic and idealistic supporters. Although not a dentist, his influence on dental education, research, journalism, literature and organization affairs is indelibly written on the pages of dental history.

The dental profession in this hour of grief must become rededicated to the advancement of all those facets of dental progress to which Dr. Gies wholeheartedly devoted the greater part of his life.

Arthur H. Merritt, President
Bissell B. Palmer, Executive Vice President

THE WILLIAM J. GIES FOUNDATION FOR THE ADVANCEMENT OF DENTISTRY, INC.

1955 Annual Report of the President

During the year 1955 the Gies Foundation made substantial progress, with promise of an increasing rate of progress in the future. Its financial condition is sound (see Addendum from Treasurer's Report). A contribution of $1,000.00 was received from the Henry Spenadel Fund, with provision for an additional contribution of $500.00 each year. This gift is for unrestricted purposes. Through Dr. William J. Gies, an anonymous donation of $500.00 was received for the capital funds of the Foundation. Also, there is knowledge of additional intended gifts.

A definite program has been set up for furthering the usefulness of the Foundation. This program has the following purposes:

I. Make the profession aware of the Foundation and its objectives. Primarily these are:

A. Establishment of a permanent, active, useful memorial to Dr. William J. Gies for his contribution to dentistry;

B. Provision for a stable organization to which persons who recognize the important role of dentistry in relation to public health and welfare, may make donations and bequests for the furtherance of dental objectives;

C. Granting of funds to assist projects related to the advancement of dentistry;

D. Filling of a void caused by the fact that there are no similar foundations;

E. Seeking additional funds to support the activities of the Foundation.

In approaching the dental profession through its dental press we shall, as frequently as possible, have publicity releases to acquaint the profession with the Foundation's program. We shall invite the dental schools and other dental institutions to make applications for grants for appropriate purposes. The primary fields of interest to the Foundation will be described. We shall also put out releases regarding the grants made by the Foundation and the related accomplishments. Also, we shall solicit funds from dental organizations and individual dentists for the support of the Foundation's work.

II. Stabilization of the Foundation's affairs.

A. With a view to broadening the activities of the Board and assuring a reserve of carefully chosen younger men available to carry on the Foundation's program in the years ahead, an Auxiliary Board has been set up consisting of six active middle-aged dentists who have shown through their activities a devotion to the advancement of dentistry. The Auxiliary Board will attend all meetings of the General Board, will become acquainted with the procedures, and will be given responsibility in relation to developing the usefulness of the Foundation. Thus, as occasions arise, well qualified successors for present members of the Board will be available, and will be acquainted with the philosophies involved as well as with the practical administration of the affairs of the Foundation.

B. Such a stabilization program requires the services of an Assistant in the office of the Foundation, who, regardless of any changes in the Personnel of the Board, will have its program at fingertips and will have the ability to maintain it.

C. The *unrestricted, expendable* funds of the Foundation, in large measure given to the Foundation so that such an Assistant might be employed, are sufficient for the purposes. It does not seem necessary at this stage to employ a well-qualified full-time person, and it is believed that it is better to employ a well-qualified part-time person rather than a less qualified one for full time.

D. The activities of the Assistant will be under the direction of the Executive Vice-President who will devote the necessary time to thoroughly teach such Assistant the history, ideals, objectives

and program of the Foundation. He will also maintain direction of the Assistant's work. In addition, the Assistant will maintain liaison with the twenty-five members of the Foundation and, insofar as practicable, with the donors to the Foundation.

The members will be invited repeatedly to make suggestions for the interest of the Foundation, in activities that will tend to advance dentistry.

E. Regular quarterly meetings of the Board of Directors will be held for routine review of the program of the Foundation and for such modifications as may seem indicated. Special meetings will be held as needed to meet special requirements.

It is believed that, through the prompt activation of this program, the Foundation will make its influence felt on the advancement of dentistry, and will fulfill the hopes of all who have its interests at heart.

Arthur H. Merritt,
President.

ADDENDUM FROM THE TREASURER'S REPORT

ASSETS

Cash in Guaranty Trust Company	$ 3,813.15	
Cash in Baldwin Federal Savings & Loan Ass'n	1,429.88	
Petty Cash on Hand	17.08	
U. S. Government Bonds Series "F" at current redemption value (face amount $13,025.00)	12,479.50	
U. S. Treasury Bonds 2½'s due 1967-72	23,839.64	
Investments in Stocks (Cost)	37,087.65	
TOTAL ASSETS		$78,666.90

LIABILITIES

Employee Withholding Taxes Payable	18.40	
Social Security Taxes Payable	8.00	
TOTAL LIABILITIES		$ 26.40

SURPLUS ACCOUNT

January 1, 1955—Balance	75,820.42	
Excess of Income over Expenses for year ended December 31, 1955	2,820.08	
December 31, 1955—Balance		78,640.50
TOTAL LIABILITIES AND SURPLUS		$78,666.90

CONTRIBUTIONS RECEIVED

Anonymous (through Dr. William J. Gies)	$ 500.00
Spenadel Foundation	1,000.00
TOTAL	$1,500.00

GRANTS DISBURSED

Dental Health Service, Inc.	$ 400.00
School for Dental & Oral Surgery, Columbia Univ.	500.00
Journal of Periodontology	200.00
TOTAL	$1,100.00

Alfred L. Kohn,
Treasurer

Announcements • • • •

ACADEMY OF PERIODONTOLOGY TO MEET SEPTEMBER 27-29, 1956

The next annual meeting of the American Academy of Periodontology will be held in the Claridge Hotel in Atlantic City, New Jersey, September 27, 28 and 29, 1956. The committee in charge of planning this meeting have organized a program and series of social events which should prove to be instructive and entertaining to all dentists and particularly those interested in periodontology. The program is as follows:

Theme: PREVENTION

Thursday, September 27

11:00 A.M.	Etiology of Periodontal Disease	Henry M. Goldman, D.M.D.
12:00 Noon	Lunch	
2:00 P.M.	Therapeutic Root Planing	Frank M. Wentz, D.D.S., Ph.D.
	Discussion Opened by	Frederick J. Halik, D.D.S.
3:00 P.M.	Subgingival Curettage	Erwin M. Schaffer, D.D.S.
	Discussion Opened by	Abram Cohen, D.D.S.
4:00 P.M.	Wound Healing	Edward Lee Howe, M.D.

Friday, September 28

9:00 A.M. to 12:00 Noon	Surgical Approach to the Periodontal Problem (25 minutes per speaker)	
	Procedure for Gingivectomy	Arnold A. Ariaudo, D.D.S.
	Procedure for Osseous Resection	Clifford Ochsenbein, D.D.S.
	Procedure for Gingivectomy and Osteoplasty	Gerald Kramer, D.M.D.
	Procedure for Reduction of the Mucobuccal Fold and Frenii	Alvin C. Hileman, D.M.D.
	Procedure for the Management of Bifurcation Problems	Frank G. Everett, M.S., D.M.D., M.D.
12:00 Noon	Lunch—Round Table Luncheon	Wilton M. Krogman, P.H.D.
2:00 P.M.	The Occlusal Problem in Periodontics	Robert L. Reeves, D.D.S.
3:00 P.M.	Orthodontics in Periodontics	John A. Kollar, D.D.S.
	Discussion Opened by	Jerome H. Skaroff, D.D.S.
4:00 P.M.	Splinting in Periodontics	Leonard S. Hirschfeld, D.D.S.

Saturday, September 29

9:00 A.M. to 11:00 A.M.	Case Planning	Saul Schluger, D.D.S. B. O. A. Thomas, D.D.S., Ph.D. Charles H. Williams, D.D.S. Jack Alloy, D.D.S.

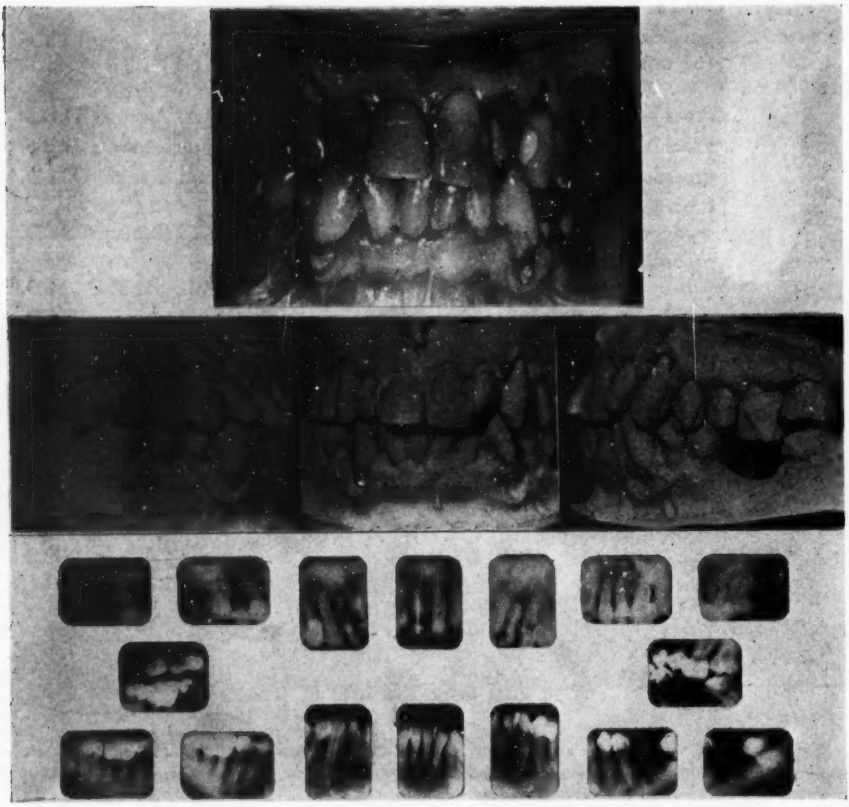
History of Case to be Discussed by Panel

A 49-year-old white male complained of marked gingival bleeding and pain on the upper left molar region. The past history revealed that he had had routine dentistry, visiting the dentist at regular intervals. Certain teeth had been extracted because of "abscesses," presumably of pulpal and periapical nature. The history also revealed that gingival bleeding had been present for many years.

Examination revealed the maxillary right third and second molars and second premolar missing, as well as the maxillary left third molar. Also missing were the mandibular left first and second molars. There was a fixed partial prosthesis in the upper right side composed of a ¾ veneer crown on the first premolar, a tru-pontic resting in a rest in an inlay in the first molar. The lower right third molar was impacted.

Examination further revealed marked gingival fibrosis, recession, bleeding to probe, a purulent exudate, and severe retraction of the gingival tissue in several areas. Caries were prevalent. The upper right central incisor was root-filled and was repaired by a crown; marked caries of the root was noted. The maxillary right lateral incisor had been treated by pulpotomy; a bridge of dentin could be discerned radiographically. The mobility pattern varied from 1 to 2-plus, being more pronounced in the maxillary left and mandibular interior regions. Pulp testing revealed all the teeth to be vital except for the maxillary right central and maxillary left lateral incisors, the root-filled teeth. The latter tooth had a widening of the periodontal membrane space periapically, as seen in the radiograph.

Examination disclosed pockets of varying depth—shallow (3mm) for the most part



but extremely deep (7mm) in several instances. In the cuspid regions the pocket base on the labial aspect was situated in the alveolar mucosa. A deep crater was also found between the maxillary left molars. Examination further disclosed marked tooth abrasion and faulty contour of the teeth.

The dental arches, although well formed, were constricted in the first premolar region. Crowding of the teeth was noticed in the lower anterior area. A discrepancy in centric relation was noticed in the examination of the occlusion, and a hit-and-glide phenomenon was easily observed. Questioning the patient concerning a habit pattern disclosed a history of bruxism and clamping, especially the latter. The patient found his jaws locked together upon rising in the morning. The prematurity was found to be in the maxillary left first premolar region and the opposing contact.

Medical history of the patient was negative.

11:00 A.M. to 11:30 A.M. Summation ..... Robert Kesel, D.D.S., M.S.

2:00 P.M. to 4:00 P.M. Clinics

1. "Vaccine Therapy in Periodontal Disease"
   H. J. V. Goldberg, L. Goldberg, and B. G. Bibby
   Eastman Dental Dispensary, Rochester, New York
2. "The Infrabony Pocket: Diagnostic Criteria for Success"
   A series of cases will be presented in chronological order of their attempted treatment with success and failure.
   John F. Prichard
   Fort Worth, Texas
3. "Periodontal Prosthesis: Diagnosis and Fabrication"
   Jack Bailin
   South Norwalk, Connecticut
4. "Post-Graduate Periodontology—1956—University of Pennsylvania"
   Teaching material and methods.
   Henry M. Goldman and Staff
5. "What Causes Bleeding Gums"
   A kinescope presentation for lay education.
   Abram Cohen
   Philadelphia, Pennsylvania
6. "A Rational Approach to Periodontal Surgery"
   Melvin Norris
   New York, New York

Subcommittees for planning the scientific sessions for the annual meeting are:

Local Arrangements

Craft A. Hopper, Chairman
Delbert P. Nachazel
Saul Schluger
Frank Paul
Francis J. Fabrizio
Marion J. Calbeck
Lester B. Older
Arvin Mann

Program

Delbert P. Nachazel, Chairman
Balint J. Orban
Henry Goldman
Albert G. Paulsen
Erwin M. Schaffer

Clinic

Saul Schluger, Chairman
William A. Themann
Robert Gottsegen
Nathan Friedman
Richard Chace
Isadore Weinimann

Entertainment

Frank Paul, Chairman
H. Roger Turner
Floyd E. Lytle
Bernard N. Walker

Publicity

Francis J. Fabrizio, Chairman
Neal Chilton
Howard Hartman
George F. Kirkland, Jr.

Registration

Marion J. Calbeck, Chairman
Ernest L. Banks
Joseph Barkley
C. Mark Gilson

Properties

Lester B. Older, Chairman
Zar Reader
David A. Hagerman
Wallace Mayo

Ladies Activities

Mrs. Robert Kesel
Mrs. Stanley Baker
Mrs. Harold Ray
Mrs. Clarke Chamberlain
Mrs. E. A. Archer
Mrs. Delbert Nachazel
Mrs. Frank Paul
Mrs. Saul Schluger
Mrs. Francis Fabrizio
Mrs. Marion Calbeck

Round Table Luncheon

Justin Towner
Frank E. Beube

TWELFTH INTERNATIONAL DENTAL CONGRESS OF THE "FEDERATION DENTAIRE INTERNATIONALE"

(International Dental Federation)

The largest and most important International Dental Meeting to be held in Roma (Italy), 7th to 14th of September, 1957.

Don't miss this splendid opportunity of meeting colleagues and friends from all over the world!

Enjoy yourselves with your wives and children with a splendid trip to Italy! Visit Rome, the Capital of the ancient civilized world! If you have never been to Italy, this is a unic occasion that you shouldn't miss; if you have been before, come once again!

You're always welcome! For information apply to: Dr. Pio Lalli, Segretario Generale del Congresso, via Boezio, 16, Roma, Italy, or to: Dr. Obed H. Moen, National Treasurer of F.D.I., 6 Main Street, Watertown, Wisc.

UNIVERSITY OF MINNESOTA

The University of Minnesota School of Dentistry and the Center for Continuation Study announces its Eleventh Annual Seminar for the Study and Practice of Oral Medicine. Sessions will be held at the Continuation Center in Minneapolis on September 28 and 29, 1956.

Further details and information may be obtained by writing to the Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces the following Postgraduate Refresher Courses:

DPG. 601—A Participation Course in Periodontics
Ten Mondays beginning Oct. 8, 1956
Tuition $250.00 Class limited to 12
Drs. Irving Glickman, Edward T. Fischer, Arthur Gold

Part of each session will be devoted to clinical practice. By treating patients each week, participants are able to carry out treatment on a number of patients and evaluate the results. The remainder of the curriculum consists of lectures and seminars devoted to the theoretical and didactic aspects of periodontology. The histopathology of periodontal disease and the "Bone Factor" concept will be presented. The clinical management of periodontal disease is analyzed in detail with special consideration given to various aspects of diagnosis, determination of prognosis and treatment planning.

DPG. 601 (A)—Participation Course in Periodontology
Monday through Friday for One Week from
October 8 through 12, 1956
Tuition $150.00 Class limited to 12
Dr. Irving Glickman and Staff

Half of the time of the course is devoted to clinical practice by the participants. The remainder of the curriculum consists of lectures and seminars devoted to the theoretical and didactic aspects of periodontology. The histopathology of periodontal disease and the "Bone Factor" concept will be presented. The clinical management of periodontal disease is analyzed in detail with special consideration given to the various aspects of diagnosis, determination of prognosis, treatment planning, provisional splinting, and the gingivectomy technique.

DPG. 602—Participation Course in Periodontology
Monday through Friday for One Week from
April 8 through 12, 1957
Tuition $150.00 Class limited to 12
Dr. Irving Glickman and Staff

This course consists of the same content as DPG. 601. It is also to be offered April 8-12, 1957 (DPG. 603).

DPG 704—Occluso-Rehabilitation I—Lecture and Seminar Course
Friday through Wednesday, 9 a.m. to 5 p.m.
Sept. 21 through Sept. 26, 1956—Classes suspended Sept. 23, 1956
Tuition $250.00
Dr. Louis Alexander Cohn

DPG 705—Occluso-Rehabilitation II—Special Participation Course
Every other week on Friday and Saturday 9 a.m. to 5 p.m.
Seven months beginning October 5, 1956
Tuition $1000.00—Enrollment limited to twelve
Dr. Louis Alexander Cohn

This is a participation course in which each student will start and complete a patient who requires all phases of Occluso-Rehabilitation. Dr. Cohn will personally supervise all the clinical and laboratory activities. The inter-relationship of Occluso-Rehabilitation and Periodontal Therapy will be stressed. It is recommended that each participant bring a patient from his private practice. However, clinic patients will be available. Occluso-Rehabilitation I is a prerequisite for this course.

For further details, write the Director of the Division of Graduate and Post Graduate Studies, 136 Harrison Street, Boston, Massachusetts.

ANNUAL MIDWEST SEMINAR OF DENTAL MEDICINE

The Midwest Seminar of Dental Medicine announces its Ninth Annual Seminar, to be held September 9-13, 1956, at Maxwelton Braes, Baileys Harbor, Wisconsin. This resort is situated in picturesque Door County, and is taken over exclusively by the seminar for that period.

This seminar has evolved in response to the growing interest of the dental profession in informal study and discussion of biologic problems which are basic to an intelligent understanding of the health and disease of the oral structures and their treatment.

Instruction and discussion will be confined to the forenoons and evenings. The afternoons will be free from any schedule and available for relaxation and rest or for informal discussion with your faculty, according to the individual desires of each member of the seminar.

The theme of our 1956 program will be the Biology of Oral Mucosa and Skin in Health and Disease.

The program will be presented in lectures, informal discussions and round table panels, under the guidance of the leading authorities listed below. Our faculty will give careful consideration to applications in clinical practice.

Stephen Rothman, M.D., Professor of Dermatology, and Head of the Department, University of Chicago School of Medicine.

Frank M. Wentz, D.D.S., Ph.D., Associate Professor of Periodontics, Loyola University School of Dentistry.

Balint Orban, M.D., D.D.S., Professor of Periodontics, and Head of the Department, Loyola University School of Dentistry and Director of the Colorado Dental Foundation.

Thomas Kirk Cureton, Ph.D., Director of Physical Fitness Laboratory, University of Illinois.

Address all correspondence to Dr. Isaac Schour, Executive Director, 808 South Wood Street, Chicago 12, Illinois.

THE AMERICAN INSTITUTE OF DENTAL MEDICINE MEETS NOVEMBER 4-8

The Next Annual Meeting of the Institute will take place at El Mirador, Palm Springs, California, November 4 to 8, 1956. The faculty will consist of:

Dr. Francis L. Chamberlain, Associate Clinical Professor of Medicine, the University of California, San Francisco.

Dr. Milton B. Engel, Associate Professor of Orthodontics, the University of Illinois, College of Dentistry.

Dr. Donald A. Kerr, Professor of Oral Pathology and Periodontia at the University of Michigan.

Dr. Helmut A. Zander, Professor of Periodontology, the University of Minnesota.

Dr. S. I. Hayakawa, San Francisco State College.

Dr. Gustav Korkhaus, the Dean of the Medical and Dental faculties of the University of Bonn on the Rhein (an outstanding investigator in the field of growth and development).

All Seminar lecturers will participate in a round table forum discussing the application of their subject to the practice of Dental Medicine. Because of the increasing interest in this annual meeting of the Institute, early registration is indicated. Applications and full information may be secured from the Executive Secretary, Miss Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

BETH ISRAEL HOSPITAL

The Dental Department of the Beth Israel Hospital announces two post graduate courses in Periodontia to be given in the Fall of 1956.

A five-day course in Occlusal Adjustment will be given from September 10 through September 14. This course will include background material on the anatomy, histology, and physiology of the Periodontium and dental apparatus. Diagnostic methods and treatment procedures for occlusal adjustment will be thoroughly discussed and demonstrated in the clinic. Participants will be limited to twelve. The tuition fee is $200.00.

A two-week seminar in Periodontia is scheduled for November 5 to November 16. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00.

Co-Directors of the Courses are Henry M. Goldman, D.M.D., Bernard Chaikin, D.M.D.

For further information, write to Director of Public Relations and Education, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

Letters to the Editor . . . .

An article published in the Journal (Carranza, Fermin A. and Carranza, Fermin A. Jr.: The Management of the Alveolar Bone in the Treatment of the Periodontal Pocket. J. Periodontology 27:29-35, Jan. 1956) has an illustration (Fig. 12) labeled Radiographic Demonstration of a Case of Bone Regeneration of the Crest—before and after. The radiograph labeled before shows an intrabony pocket on mesial of the right central incisor. The radiograph labeled after shows no intrabony pocket. The radiograph labeled before is a right diagonal view and includes the right cuspid tooth. The right central incisor measures 28 millimeters in length. The radiograph labeled after is a left diagonal view and includes the left cuspid tooth. Here the right central incisor measures 26 millimeters in length. It is obvious that the radiographs labeled before and after have been made at angulations that vary about 20 degrees mesio-distally. The before radiograph would tend to widen the intrabony pocket and make it more pronounced. The after radiograph would tend to throw the bone mesial to left central incisor on to the mesial surface of the right central incisor. The difference of 2 millimeters in length of the right central incisor indicates that the right central incisor in the after radiograph has been fore-shortened. Again the after radiograph would tend to decrease the depth of the intrabony pocket.

If one uses radiographs before and after treatment to compare bone levels, one must attempt to duplicate the original radiograph. The identical film packet placement and mesio-distal and vertico-horizontal angulations should be used. Similarly the film exposure and developing times should be repeated so that radiopacities and translucencies do not vary. Differences in angulation as well as opacity of radiographs open to question the validity of the results.

Harold R. Schreiber, D.D.S.
Washington University School of Dentistry
4559 Scott Ave., St. Louis, Mo.

To the Editor:

I am writing to you in your capacity as Editor of the Journal of Periodontology to suggest a more appropriate name for a comparatively new surgical periodontal procedure which has become quite popular in recent years. This procedure is employed to deepen the space between the turn of the tissue in the vestibule of the mouth and the gingival margin around one or more teeth.[1,2,3,4]

At this moment, such names as frenectomy, frenotomy and mucobuccal fold extension are being used to denote this procedure. The first two terms actually describe only the procedure in which a frenum is involved. Quite often it is more than just a frenum—a whole area may be involved. The procedure employed is actually a plastic surgical one in the vestibule of the mouth. It is a reshaping of the vestibule. Just as we now use

1. Gottsegen, R.: Frenum Position and Vestibule Depth in Relation to Gingival Health. O. Surg., O. Med., and O. Path. 7:1069, 1954.

2. Stewart, J. M.: Reattachment of Vestibular Mucosa as an Aid in Periodontal Therapy. J.A.D.A. Sept. 1954, p. 283.

3. Nabers, C. L.: Repositioning the Attached Gingiva. J. Period. 25:138, 1954.

4. Goldman, H., Fox, L. & Schluger, S.: Periodontal Therapy, C. V. Mosby Co., St. Louis, 1956.

gingivoplasty to denote reshaping of the gingiva, and osteoplasty to denote reshaping of the alveolar bone, I would like to submit the word "vestibuloplasty" to denote the reshaping of the oral vestibule. I have been using this term at Temple University where it has been helpful in conveying the image of the procedure to the students.

I hope that you will see fit to publish this letter in the Journal so that the members of the Academy can read and comment on it and perhaps may decide to adopt this suggestion.

Neal W. Chilton, D.D.S.
225 West State Street
Trenton 8, New Jersey

To the Editor:

In a letter to the editor published in the January 1956 issue of the Journal of Periodontology Dr. F. A. Carranza, Jr. stated that I was in error when I credited Dr. Saul Schluger with the conception of the principle of osseous resection. He maintained that it was his father, Dr. F. A. Carranza, Sr. who first presented this principle. I do not think we were referring to the same "principle."

The quotation from my paper that Carranza, Jr. used, "The principle of osseous resection was conceived and presented by Schluger" was only part of the opening sentence. The complete sentence and the subsequent one are as follows: "The principle of osseous resection was conceived and presented by Schluger to overcome a serious shortcoming in existing periodontal surgical techniques in certain aspects of case management. This principle grew out of repeated failures to maintain the results achieved by gingivectomy."

The principle referred to was not the mere removal of bone to eliminate a pocket. I was referring to recognition of the principle of the interdependence of the soft and hard tissue anatomically and physiologically, the difference in their behavior in periodontal disease, and the need to take this into account in order to achieve and maintain a successful result. Embodied in this principle is the realization that failures in therapy were due, on occasion, to a lack of awareness that the gingival and osseous architecture must be harmonious. Successful periodontal surgery cannot be the mere removal of tissue with no consideration of form and function. All this and more was implied in the use of the term "principle" in the opening statement of my paper.

There was no intent to ignore the senior Dr. Carranza's contribution to the periodontal literature. I had not read his papers in the original, but I did read the material abstracted from them in Glickman's "Periodontology." There was nothing in this material that set up the rational concept mentioned above, but rather it presented an effective method of dealing with some types of infrabony pockets. Indeed, even in the paper written by both Carranzas in the January 1956 issue of the Journal of Periodontology no mention is made of this principle, but once again a technical approach to the elimination of some types of "intra-alveolar pockets" is presented.

It might very well be of importance to set the record straight in matters of this kind. I hope this exchange of letters will clear up any misconceptions that Dr. Carranza Jr. might have, and correct any erroneous impression I might have created.

Nathan Friedman, D.D.S.
Beverly Hills, California

To the Editor:

When the article "Effectiveness of Berliner Epithelial Scalpel in Removing Epithelial Lining of Periodontal Pockets" by Dr. F. W. Wertheimer first appeared in the Journal of Periodontology (Vol. 25:264-271) October, 1954, I felt no comment was necessary since the basis for critically evaluating any instrument, designed for the purpose described by Dr. Wertheimer was not given in the paper.

However, I have recently become aware of reports concerning the inaccuracy in the effectiveness of the instrument I have designed. Since this conclusion of inaccuracy rests on methodologic weaknesses, the evaluation of any other instrument designed for the same purpose may be subjected to a similar noncritical evaluation. I therefore feel it incumbent to point out what must be considered to be basic inaccuracies in the conclusions reached by Dr. Wertheimer.

Two very definite points stand out. There is no random selection of the data, admitted by Dr. Wertheimer, which I feel, should be accepted as a disarming admission. The confusing inadequate definition of the use of "chance" employed descriptively, makes the scientific validity of the findings questionable.

The data of 16 and 14 "cases" are all lumped together. No breakdown appears of how many "cases" showed inflammation (in terms of acute or chronic—as related to edema) and how many did not. One must question how distribution would affect the final conclusions.

Dr. Wertheimer's evaluation concerning the "chance for achieving complete removal of pocket epithelium" is based on a simple frequency distribution of the number examined for this purpose; this result was then multiplied by the factor of 100. The use of the word "chance" here, is based on the frequency distribution existing in a nongraded population. To the unwary clinician, i.e., a 14% success might easily imply that only 14 out of every 100 patients may be expected to benefit (without any selection of the patients).

In the simple frequency distribution employed by Dr. Wertheimer, it is possible for all 86 slides showing failure of completeness in removal to be derived from a very small number of cases. For example, comparing the number of good and complete results as against the number or fair, poor and no results in cases 2 and 3 of the article, we see that only 6 slides showed "good" and 20 were "fair" or "worse." This is an incidence of 6/26 x 100 or approximately 23% good or better "success" as defined by Dr. Wertheimer, but in reality is a 50% "success" as far as the number of patients is concerned, since 19 of the 20 fair or poorer results are derived from one patient and the 6 good or better results, from the other. Such a breakdown is necessary in critical evaluation.

In view of these comments, I feel that no conclusions can be drawn as to the effectiveness of not only the Berliner Epithelial Scalpel, but any other instrument designed to be used for a similar purpose.

Abraham Berliner, D.M.D.
30 Central Park South
New York 19, New York